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Understanding the Role of Platelet Concentrates in Dentistry: A Review

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ABSTRACT

Platelets are the first to appear at the site of injury and play a crucial role in hemostasis and wound injury. They not only help in repairing the tissue but also regenerate them. Whole blood can be centrifugated at different speed and time to form variable types of platelet concentrates with or without anticoagulant like Platelet rich plasma (PRP), Platelet rich fibrin (PRF). These concentrates are being universally used in various fields of medicine and dentistry to improve wound healing, regenerate tissues, assist in early healing and and serve as substititute for bone graft. Their activity against microbes had evolved them as antimicrobial agent as well. This article provides complete insight into background, evolution, generations, mechanism of action, recent advances and uses of platelet concentrates into various fields of Dentistry.

Keywords: Platelet Concentrates, Platelet Rich Plasma, Growth Factors, Wound Healing.

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INTRODUCTION

The major goal of health sciences in 21st century is to develop clinically relevant strategies for tissue generation The reason for the realization is that the best substitute of an organ/tissue lost is the actual organ/tissue itself. Blood is a mixture of cellular elements, colloids and crystalloids each having different specific gravity with regenerative capabilities. In increasing order, the specific gravity of blood components is plasma, platelets, leucocytes (Buffy Coat [BC]) and packed red blood cells (PRBCs). Each component has specific count and function. Human platelet count is 200000 ± 75000/mL with a half-life of 7-10 days. Upon contact with exposed endothelium (due to damage tissue or wound) the platelets get activated and are known to release key wound healing factors namely Platelet derived growth factor (PDGF), vascular endothelial growth factor (VEGF), transforming growth factor (TGF) and epidermal growth factor (EGF). Platelets begin to actively secrete these proteins within 10 min after clotting, with more than 95% of the pre- synthesized growth factors secreted within 1 hour. For the balance of their life (5 to 10 days), the platelets synthesize and secrete additional proteins. As the direct platelet influence begins to subside, macrophages (stimulated by the platelets) take over the responsibility for wound-healing regulation by secreting their own factors. Thus, the platelets are ultimately initiator for wound repair.

Numerous studies have emphasized that human platelets are a good source of antimicrobial peptides such as: Thymosin b4, platelet basic protein, platelet factor 4, connective tissue activating peptide Ⅲ, fibrino-peptides A and B and chemokine (C-C motif) ligand 51. There are special receptors on the platelets that are known to aggregate with bacteria. Platelets also participate in generating oxygen metabolites, including hydrogen peroxide, superoxide, and hydroxyl free radicals.[2] Largely, platelets demonstrate first line activities against the blood-borne pathogens and also play an important role in the innate host defense against the initiation and progression of infections.[2] In fact Garraud et al,[3] in 2015 claimed that "platelets are innate and inflammatory cells and do not only assist immunity but are immune cells themselves".

In Dentistry, introduction of platelet concentrates have not only assisted in early wound healing but also have shown promising results in regenerative procedures. Since, it is the most cost effective method in regenerating bone and pulpal tissues it

The present review highlights the composition, historical background, mechanism of action, preparation and clinical implications of platelet concentrates in the fields of Dentistry.

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History

Kinsley in 1954 first gave the term PRP for thrombolytic concentrate. In late 1970s, Matras introduced 'fibrin glue' also known as fibrin sealants or fibrin tissue adhesives. There are two types of fibrin sealants: Homologous and autologous. Homologous/commercial variant was prepared by mixing 2 components, i.e., fibrinogen component containing factor XIII and the thrombin component containing calcium ions. Homologous fibrinogen concentrates were prepared from plasma cryoprecipitate or from Cohn fraction. However, it has risk of transmitting infections hence, autogenous fibrin are preferred which comprise of fibrinogen and clotted serum which contains thrombin as end product. In 1986, Knighton et al,[4] first demonstrated that PC successfully promote healing and they termed it as "platelet-derived wound healing factors (PDWHF)", which was successfully tested for the management of skin ulcers. Whitman et al,[5] (1997) named their PRP as "platelet gel". All these products were designated as PRP without knowing quality of its content or architecture, and this paucity of terminology continued for many years. Some commercial companies, in lieu of better visibility, started labeling their products with distinct commercial names for example: P-PRP was commercialized by the name Vivostat PRF (Alleroed, Denmark). However, as the name implies it is not a PRF but produces a PRP product.

Choukroun J et al,[6] in France (2006) developed another form of platelet concentrates which was self-clotted and termed "Platelet-Rich Fibrin (PRF)" due to the strong fibrin gel polymerization. All these preparations prior to this required extrinsic clotting factor to initiate the clotting pathways and were considered the 'First Generation' of platelet concentrates or PRPs while the preparation which does not require extrinsic clotting factors to initiate clotting were considered 'second generation' of platelet concentrates or PRFs.

Evolution of new terminology appeared when Dohan Ehrenfest et al,[7] pointed out that the PC were also associated with various forms of circulating cells, particularly leukocytes, and labeled it as L-PRP (Leukocyte rich platelet rich plasma).

Recent advances in Platelet concentrates

Sacco (2006) introduced "Concentrated Growth Factors (CGF)".[8] A special centrifuge called Medifuge (Italy), is used to prepare CGF.

A concept of fabricating growth factors-enriched bone graft matrix (also known as "sticky bone") using autologous fibrin glue has been demonstrated since 2010.[9] Sticky bone provides stabilization of bone graft in the defect, and therefore, accelerates tissue healing and minimizes bone loss during healing period.

Mourão et al,[10] (2015) described a technique to obtain an injectable form of PRF called i-PRF. This could be injected or mixed with bone graft to give a well agglutinated "steak" for bone grafting.

Tunalı et al,[11] in 2014, introduced a new product called T-PRF (Titanium prepared PRF). The use of titanium tubes for collection and centrifugation instead of glass tubes was established on the hypothesis that Titanium may be a more efficient platelet activator than silica, for preparing L-PRF [Table 1].

Classification

The first classification about platelet concentrate was proposed by Dohan Ehrenfest et al,[12] [Table 2] in 2009. Later in 2012, Mishra et al,[13] proposed another classification which was limited to PRP and applicable to sports medicine only.

At about the same time DeLong et al,[14] introduced another classification system called PAW (Platelets quantity, Activation mode, White cells presence). However it again was only restricted to PRP families and was similar to classification by Mishra et al.

Process of preparation

Gabriela Martin et al,[15] introduced single step centrifugation preparation technique. 10 ml of whole venous blood is drawn from patient's arm and equally divided into 4, 2.5ml tubes to obtain 4.5-5ml of PRP. Each tube contains 3.8% sodium citrate to prevent blood coagulation. Blood is centrifugated at 1500rpm for 30 minutes to separate the precipitated erythrocytes from PRP suspension. The plasma next to the precipitated erythrocytes, which is rich in platelets, is aspired into a syringe and placed into 2, 3.6ml vials. Fibrin coagulation of PRP is initiated by adding calcium chloride (0.025mol/L). For 2.5ml PRP, 1.25 ml $CaCl_2$ is added into each tube and the tube is shaken by hand. Fibrin matrix formation occurs within 10 minutes after the addition of $CaCl_2$. The solution of PRP and $CaCl_2$ is immediately aspirated into an insulin syringe and injected into the desired area.

In two step centrifugation, 20ml of venous blood is drawn and transferred to tubes containing anticoagulant to avoid platelet activation and degranulation. The tubes are then centrifugated at 1000g for 10 minutes (soft spin). 3 distinct layers are formed from top to bottom platelet poor plasma (PPP), an intermediate layer and buffy coat characterized by an increase in platelet concentration. Majority of top layer is discarded and the remaining content is transferred to an empty tube and centrifugated for 15 minutes at 2200g (Hard spin).Concentrated PRP is obtained by discarding the majority of the remaining platelet poor plasma with equal volumes of sterile saline solution containing 10% calcium chloride and sterile bovine thrombin (100U/ml) in a sterile well plate.

The classical technique for preparation of PRF was developed by Dr. Choukroun,[16] in 2000. The technique has been authorized by the French Health

Ministry. The equipments required for the technique include PC-02 table centrifuge and a blood collection kit consisting of a 24 gauge butterfly needle and 9ml blood collection tubes. A sample of blood is collected from patient without anticoagulant in 10 ml tubes which are immediately centrifuged at a rate of 3000 rpm for 10 minutes. During the centrifugation process, when the blood gets in contact with the test tube wall of the platelet gets activated leading to the initiation of coagulation cascade. The resultant product consist of three layers from top to bottom PPP, PRF in the middle and RBCs at the bottom of the test tube. The fibrin clot obtained is removed from the test tube after centrifugation. It can be formed into a membrane by squeezing out the fluids present in the fibrin clot and placed in the desired area.

Mechanism of action [Figure 1]

The platelets contained in this concentrate of autologous plasma release their alpha granules after the coagulation process has been locally trigged in the wound site. These alpha granules contain a cocktail of growth factors which promote proliferation, chemotaxis and the differentiation of cells, which are essential to osteogenesis. Thus, besides its procoagulant effect, PRP is a source of growth factors involved in initiating and sustaining wound healing by accelerating bone repair, promoting fibroblast proliferation, and increasing tissue vascularity.[17] Platelet-rich plasma gel is formed by mixing PRP (derived from the centrifugation of autologous whole blood) with thrombin and calcium chloride. Adding thrombin and calcium chloride to PRP automatically activates the alpha granules to release the following biological growth factors: platelet-derived growth factor (PDGF), transforming growth factor-beta (TGF-b), vascular endothelial growth factor (VEGF), insulin-like growth factor I, epidermal growth factor (EGF) and epithelial cell growth factor.[18,19]

Other cytokines released by PRP alfa granules are TGF-b1 and TGF-b2, both of which are involved in connective tissue repair and bone regeneration. Their most important role appears to be to stimulate fibroblast chemotaxis and the production of collagen and fibronectin by cells while inhibiting collagen degradation by decreasing proteases and increasing protease inhibitors.

Applications in Dentistry

Periodontics

Generally PRP and PRF are used in Periodontology for treatment of infrabony defect. Aroca et al,[20] in a randomized clinical trial concluded that addition of a PRF membrane placed under the MCAF (modified coronally advanced flap) provided additional gain in gingival/ mucosal thickness but inferior root coverage over 6 months follow up period compared to the conventional therapy.

Eren and Atilla,[21] in 2012 treated bilateral gingival recession with (CAF) coronally advanced flap and (SCTG) subepithelial connective tissue graft on one side and CAF with PRF on other side. They found improvement in all parameters with both the techniques. Since use of PRF was practical and simple to perform and also eliminates the requirement of donor site wound, they suggested that CAF + PRF as a better alternative to CAF + SCTG.

Edos,[22] (2016) have highlighted the inconsistent results of PRF in covering Miller Class I and Class II gingival recessions with no improvement in terms of root coverage, keratinized mucosa width, or clinical attachment level, but it was shown to have increased the gingival thickness and had better wound healing compared to other treatment modalities.

Implant surgery

PRF plays a vital role in implant surgery by the accelerating the cicatricial process of bones and gums in dental surgeries, especially in dental implants. It has high potential for tissue regeneration, capability of transforming adult stem cells into specific cells for the formation of bone and gingival tissue, ability to regenerate the tissue vascularization network and thus the need to remove bone from another part of the body for bone grafting may not be required, making the procedure more comfortable for the patient as well as dentist.[23,24] Replacing hopeless tooth with an immediate implant and PRF placement potentially improves implant bed preparation and motivates osseointegration.[25] Anitua (2006) showed that the osseointegration of implants was enhanced by coating the implant surface with PRP prior to insertion into the alveolus.[26]

Oral surgery

Extractions and impaction in oral surgery are the most common procedure in dentistry. These procedures are most often associated with post-operative pain, swelling, dry socket and trismus. Moreover, patients on anticoagulant therapy experience prolonged bleeding after tooth extraction. These common problems have been dealt with different therapies like LASER therapy to reduce infection and thus post-operative pain after extraction, Fibrin sponge to decrease bleeding and hence, assist in clot formation or prevent dry socket. Recently, PRP/PRF are preferred because of being economical, osteoinductive propertities, promote growth factors to induce early healing.

Alissa et al,[27] in 2010,conducted a pilot study on the effect of PRP on healing of the hard and soft tissues of extraction sockets. The study concluded that soft tissue healing was significantly improved in patients treated with PRP compared with patients of the control group (no treatment). Moreover, patients untreated with PRP experienced complications (dry sockets and acutely inflamed alveolus), which were considered to be borderline statistically significant. Radiographic evaluation revealed a statistically

significant difference only for sockets with a dense homogeneous trabecular pattern. Of interest, Alissa et al. (2010) also analyzed the post-operative pain of patients of the two groups (treated and untreated) and they reported significantly more pain in the control group, especially in the first three days post intervention. Similar findings have been reported by Ruktowski et al,[28] (2010) who used digital radiography and Computer Tomography (CT) scan analysis to track changes in radiographic density at PRP- treated sites in comparison to ipsilateral not-PRP treated sites. The PRP- treated sites demonstrated early and a significant increased radiographic density over baseline measurements following tooth removal. The greatest benefit attributed to PRP is during the initial 2-week post-operative healing time period: 6 weeks for control extraction sites to reach comparable bone density were required whereas PRP- treated sites achieved this at week 1. Post-operative pain and bleeding were not significantly affected by PRP application.

Daif,[29] (2012) investigated the effect of autologous PRP on bone regeneration in mandibular fractures. He concluded that direct application of the PRP along the fracture lines may enhance bone regeneration. Wojtowicz et al,[30] (2007) compared the effects of stimulating the osteogenesis of the alveolar bone by transplants of autologous bone marrow and freshly isolated mononuclear cells from bone marrow, containing CD34+ cells and PRP. It was shown that newly formed bone increased under the influence of PRP. This treatment was more effective than that using the population of CD-34 bone marrow- derived stem cells.[30]

PRF has also shown promising result in sinus lift procedures, ridge or sinus augmentation and surgical treatments of peri- implantitis defect.[31-33]

Endodontics

Cell type-specific manner makes PRF beneficial to periodontal regeneration. In addition, activation of phosphorylated extracellular signal-regulated protein kinase, osteoprotegerin, and alkaline phosphatase expression by PRF suggests the pivots for new periodontal attachment formation.[34] Zhao et al.[35] demonstrated that the combined use of PRF and cell sheet fragments of PDL stem cells or PRF alone, in replanted incisors in an animal experiment, which had an extraoral dry time of 2 hours, showed effective healing characterized by regeneration of PDL like tissues and reduction of ankylosis.

PRF is also used in regenerative Endodontics in open apex cases. Keswani et al,[36] (2013) reported that PRF serve as a potentially ideal scaffold in revascularization of immature permanent teeth with necrotic pulp as it ia rich in growth factor, enhance cellular proliferation and differentiation and act as matrix for tissue ingrowth.

Orhan et al,[37] (2012) displayed reparative dentine formation after direct pulp capping with the help of PRP in the rat dental pulp as effectively as MTA and calcium hydroxide. However, research is still going on to focus on the characteristics of PRP-promoted reparative dentine formation in wounded dental pulps.

Future research in context of platelet concentrates

- Characterization of PRP and PRF as the term are still unambiguously used
- Role of PRP and PRF in direct and indirect pulp capping procedures
- Synergistic and antagonistic relation to various bone grafts, GTR membrane

Table 1: Recent advances in PRF concentrates

Recent advances		Technology	Advantages
Concentrated Growth Factors (CGF)	Sacco (2006)[8]	Venous blood centrifugated at 2400-2700 rpm	allows the separation of a fibrin matrix which is much denser, larger and richer in growth factors.
Sticky bone	Sohn (2010)[9]	25-60cc of venous blood is centrifugated at 2400-2700rpm using a specific centrifuge (Medifuge, Silfradentsrl, Sofia, Italy) for 2 minutes. 2 layers are obtained deeper layer of RBCs and superficial layer of AFG. This AFG is extracted using syringe and mixed with particulate bone powder which is allowed to rest for 5-10 min for polymerization. The yellow colored mass thus obtained is called sticky bone	Autologous fibrin glue mixed with bone graft. provides stabilization of bone graft.
A- PRF (Advance PRF process, France)	Choukroun (2010)[16]	Venous blood centrifugated at 1300 rpm for 14 minutes	Contains more monocyte which release BMP 2 Earlier vasculaization, fast tissue growth, and more cytokines.
Titanium PRF (T-PRF)	Tunali et al (2014)[11]	Titanium tubes used for collection of blood. 9 ml venous blood collected in titanium tubes and centrifugated at 2800 rpm for 12 minutes	Immensely organized network along with a continuous integrity and fibrin network was thicker that covered a larger area.
i-PRF (injectable PRF)	Mourao et al (2015)[10]	9ml Venous blood centrifugated for 2 minutes at 3300 rpm. The orange coloured fluid obtained by the process is i-PRF	Obtained in injectable form.

Table 2: Classification of Platelet concentrates

Classification proposed by	Basis of classification	Nomenclature
Dohan Ehrenfest et al[12] (2009)	The cellular content (primarily leukocytes) and the fibrin architecture	1.Pure platelet-rich plasma (P-PRP) or Leukocyte poor platelet rich plasma (LP-PRP) 2. Leukocyte and platelet- rich plasma (L-PRP) 3. Pure PRF (P- PRF) or Leucocyte- poor PRF 4. Leukocyte- and platelet rich fibrin
Mishra et al [13] (2012)	Based on presence or absence of leukocytes and whether or not the PRP is activated and all types can fall into 2 sub-types: A: Platelets > 5 × baseline or B: Platelets < 5 × baseline. In all the types "solution" means non-activated PRP and gel means activated PRP.	Type 1: L-PRP solution Type 2: L-PRP gel Type 3: P-PRP solution Type 4: P-PRP gel
DeLong et al[14]]**Error! Bookmark not defined.**	classification system called PAW (Platelets quantity, Activation mode, White cells presence)	Similar to Mishra et al

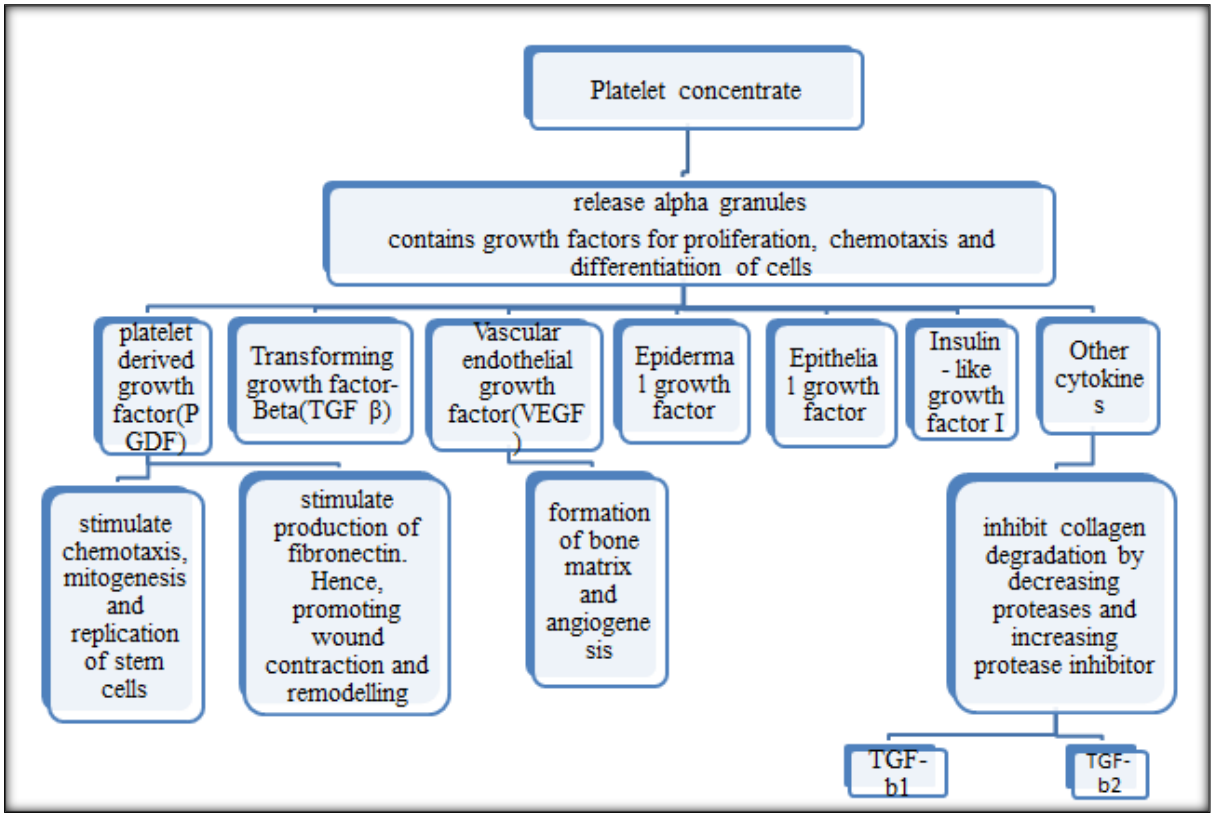

Figure 1: Mechanism of action

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