

# **Clinical Evaluation of Platelet Rich Plasma and Acellular Dermal Matrix Allograft in the Management of Gingival Recession**

Thesis

Submitted to the Faculty of Dentistry Ain Shams University In  
partial fulfillment of the requirements of master degree in Oral  
Medicine, Periodontology and Oral Diagnosis

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**Ain Shams University  
2011**

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التقييم الإكلينيكي للبرازما الغنية بالفانح الدموية  
وإعادة الطعم الإسوي  
البدى الآخلى الطوق "الأوديرم" لعلاج تراجع اللثة

رسالة مقدمة من

الطبيبة / رانيا فريد السيد احمد عوض الله

توطئة للحصول على درجة ماجستير

فى طب الفم وعلاج اللثة

كلية طب الاسنان

جامعة عين شمس

2011

## تحت اشراف

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## Summary

The treatment of buccal gingival recession is a common requirement due to aesthetic concern or root sensitivity in patients with high standards of oral hygiene. The ultimate goal of a root coverage procedure is the complete coverage of the recession defect with good appearance related to adjacent soft tissues and minimal probing depth (PD) (*Miller 1985, Roccuzzo et al. 2002, Clauser et al. 2003*). The coronally advanced flap (CAF) procedure is a very common approach for root coverage. This procedure is based on the coronal shift of the soft tissues on the exposed root surface (*Allen & Miller 1989, Pini Prato et al. 2000*). This approach may be used alone or in combination with soft tissue grafts (*Wennström & Zucchelli 1996*), acellular dermal matrix (ADM) (*Harris 1998*) and platelet-rich plasma (PRP) (*Marx et al. 1998*).

This study evaluated the clinical changes in terms of Gingival recession depth, Probing Depth (PD), Clinical Attachment Level (CAL), Width of keratinized gingiva, Plaque Index (PI), Gingival index (GI) before and after treatment of gingival recession by using the acellular dermal matrix graft or “Alloderm” & use of growth factors by use of PRP with coronally positioned flap .

The present study was designed as a clinical trial for treatment of gingival recession defects. Fourteen subjects were selected from the outpatient clinic, Department of Oral Medicine, Periodontology and Diagnosis, Ain Shams University. With an age ranged from 20 to 45 years.

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**List of abbreviation**

AAP.....	American Academy of Periodontology
ADM.....	AlloDerm
ATP.....	Adenosine triphosphate
CPF.....	Coronally positioned flap
CAL.....	Clinical Attachment Level
CEJ.....	Cementoenamel junction
CHX.....	Chlorhexidine digluconate
CPT.....	Coronally positioned tunnel
CT.....	Connective tissue
CTG.....	Connective tissue grafts
EGF.....	Epidermal growth factor
GI.....	Gingival index
GTR.....	Guided tissue regeneration
IGF-1.....	Insulin like growth factor-1
LPS.....	Lipopolysaccharid
PD.....	Probing Depth
PDAF.....	Platelet-derived angiogenesis factor
PDEGF.....	Platelet derived epidermal growth factor
PDGF.....	Platelet derived growth factor
PDLC.....	Periodontal ligament cells
PF-4.....	Platelet factor 4
PGE2.....	Prostaglandin
PI.....	Plaque index
PPP.....	Platelet poor plasma
PRP.....	Platelet Rich Plasma
RBC.....	Red blood cells
RCT.....	Randomized clinical trials
RD.....	Recession depths
SCTG.....	Subepithelial connective tissue graft
TGF- $\beta$ .....	Transforming growth factor - $\beta$
VEGF.....	Vascular endothelial growth factor

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## Acknowledgement

Thanks to God before and after. I would like to express my deepest gratitude to Prof. Dr. Khaled Abdel Ghaffar, chairman of the oral medicine and periodontology department for his kind directions and persistent supervision through this work.

I am greatly indebted to Dr. Mohamed El Mofty, Lecturer of oral medicine and periodontology, faculty of dentistry, Ain Shams University for his thorough supervision of this work and right observations.

I would like to express my deepest gratitude to Prof. Dr. Hala Kamal Previous chairman of the oral medicine and periodontology department for her support, kindness and help. I would also like to express my appreciation to all the staff of the department of oral medicine and periodontology for their sincere help and cooperation.

I would like to express my deepest gratitude to my doctors in the oral medicine and periodontology department of Tanta University specially Prof. Dr. Hoda Elgeindy & Prof. Dr. Lobna EL Gamal who taught me how wonderful and exciting the periodontology department is.

Really I couldn't find enough words to express my gratitude and appreciation to my ***mom***, dad and my sisters: ***Dr. Faten*** & Eman. I would also like to especially thank my own family : ***Captain Mahmoud*** my wonderful husband and my amazing daughter ***Lama*** from whom I took a lot of their own time to finish this study

Finally I would like to thank the patients of the study for their voluntary characters and for their valuable contribution into the medical research.

## INTRODUCTION & REVIEW OF LITERATURE

Periodontitis is a worldwide disease often resulting in severe bone loss around teeth. The most desirable goal of therapy is to achieve the regeneration of tissues destroyed by Periodontitis including alveolar bone, cementum and periodontal ligament (*Lins et al. 2003*). (*Lindhe 1989*). Esthetics is another important consideration (*Allen 1988*).

Buccal gingival recessions in the anterior region represent one of the most important challenges for periodontists. A variety of conventional surgical procedures have been described to achieve soft tissue coverage of exposed root surfaces, such as the laterally positioned flap (*Grupe & Warren 1956*), coronally positioned flap (*Harvey 1665*), free gingival graft (*Miller 1982*), subepithelial connective tissue graft (SCTG) with a coronally positioned flap (*Langer & Langer 1985*), and guided tissue regeneration (*Trombelli et al. 1994*). Few histologic studies have examined the quality of healing after root coverage (*Majzoub 2001 & Cummings 2005*). The formation of long junctional epithelium is generally expected after conventional mucogingival surgery (*Caffesse et al. 1984*). However, a limited amount of regeneration can be achieved with the conventional techniques (*Weng et al. 1998*).

Gingival recession is the exposure of the root surface resulting from migration of the gingival margin apical to the cemento-enamel junction (CEJ). It may be localized or

generalized and can be associated with one or more tooth surfaces (*Kassab and Cohen 2003*).

Gingival recession as defined by the American Academy of Periodontology is the location of the gingival margin apical to the cemento-enamel junction (*AAP 2009*). There may be several causes for recession that include mechanical factors (trauma from improper oral hygiene practices, tooth brush abrasion), inflammatory factors (poor oral hygiene, periodontal disease, restorative considerations plaque-induced inflammation, calculus), anatomical factors (minimal vestibular depth, high frenum attachment, thin periodontium, root prominence and tooth position) and/or heredity factors. The effects of these factors have been shown to contribute to sensitivity, cervical abrasion, root caries and compromised esthetics (*Tugnait & Clerehugh 2001*). It is evident that gingival recession is more prevalent in patients with periodontal diseases and smokers. This higher rate of gingival recession is in smokers compared to non-smokers. The higher rate could be due to a decrease in gingival crevicular fluid, less bleeding on probing and also fewer gingival blood vessels, which is common during smoking. The other reason could be an increase in colonization of periodontal pathogens both in shallow and deep periodontal pockets. Alteration in immune response such as altered neutrophil chemotaxis, Phagocytosis and an increase in the production of prostaglandin (PGE<sub>2</sub>) by monocytes in response to lipopolysaccharide (LPS) is also a contributing

factor. The exact changes in the immunologic mechanisms involved in the rapid tissue destruction seen in smokers are currently unclear (*Newman 2006*).

Epidemiological studies show that more than 50% of subjects in the populations studied have one or more sites with recession of at least 1 mm, buccal sites being most commonly affected. Higher levels of recession have been found in males than females (*Susin et al. 2004*). Recession at the buccal surfaces is common in populations with good oral hygiene (*Neely et al. 2005*). Whereas with poor standards of oral hygiene it may affect other tooth surfaces (*Baelum et al. 1986*). Gingival recession at the lingual surfaces of lower anterior teeth showed a strong association with the presence of supragingival and subgingival calculus (*van Palenstein Helderman et al. 1998*).

Gingival recession may occur without any symptoms it can give rise to pain from exposed dentine, patient concern about loss of the tooth, poor esthetics or root caries. The denuded root surfaces cause deterioration in the esthetic appearance, dentin hypersensitivity and inability to perform proper oral hygiene procedures (*Zucchelli et al. 2006*).

The width of the attached keratinized gingiva varies in different individuals and on different teeth of the same individual. Recession refers to the location of the gingiva and not its condition. Sites with gingival recession are more likely to