بسم الله الرحمٰن الرحيم

"إِنَّمَا يَخْشَى اللَّهَ مِنْ عِبَادِهِ الْعُلَمَاءُ"

صدق الله العظيم

سورة فاطر: آية ٢٨

THE ASSESSMENT OF OSTEOPONTIN LEVEL IN GINGIVAL CREVICULAR FLUID IN PATIENT WITH PERIODONTAL DISEASE

Thesis

Submitted For Partial Fulfillment of the Requirement of Master Degree

IN

Oral Medicine and Periodontology

By

Marwa Mohamed Tawfiq Ali Abd –Al Qader

B.D.S.

(October 6 University)

Oral Medicine and Periodontology Department
Faculty of Oral and Dental Medicine
Cairo University

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Supervisors

Professor Dr. **Mahmoud *Ibrahim El-Refaie***
Professor of Oral medicine and Periodontology
Chairman of Oral medicine , Periodontology and Diagnosis Department
Faculty of Oral and Dental medicine
Cairo University

Professor Dr. **Azza Ezz-El Arab**
Professor of Oral medicine and Periodontology
Vice dean for post graduate studies
Faculty of Oral and Dental medicine
Cairo University

Dr.**Gehan Gharib Madkour**
Lecturer of Oral medicine and Periodontology
Faculty of Oral and Dental medicine
Cairo University

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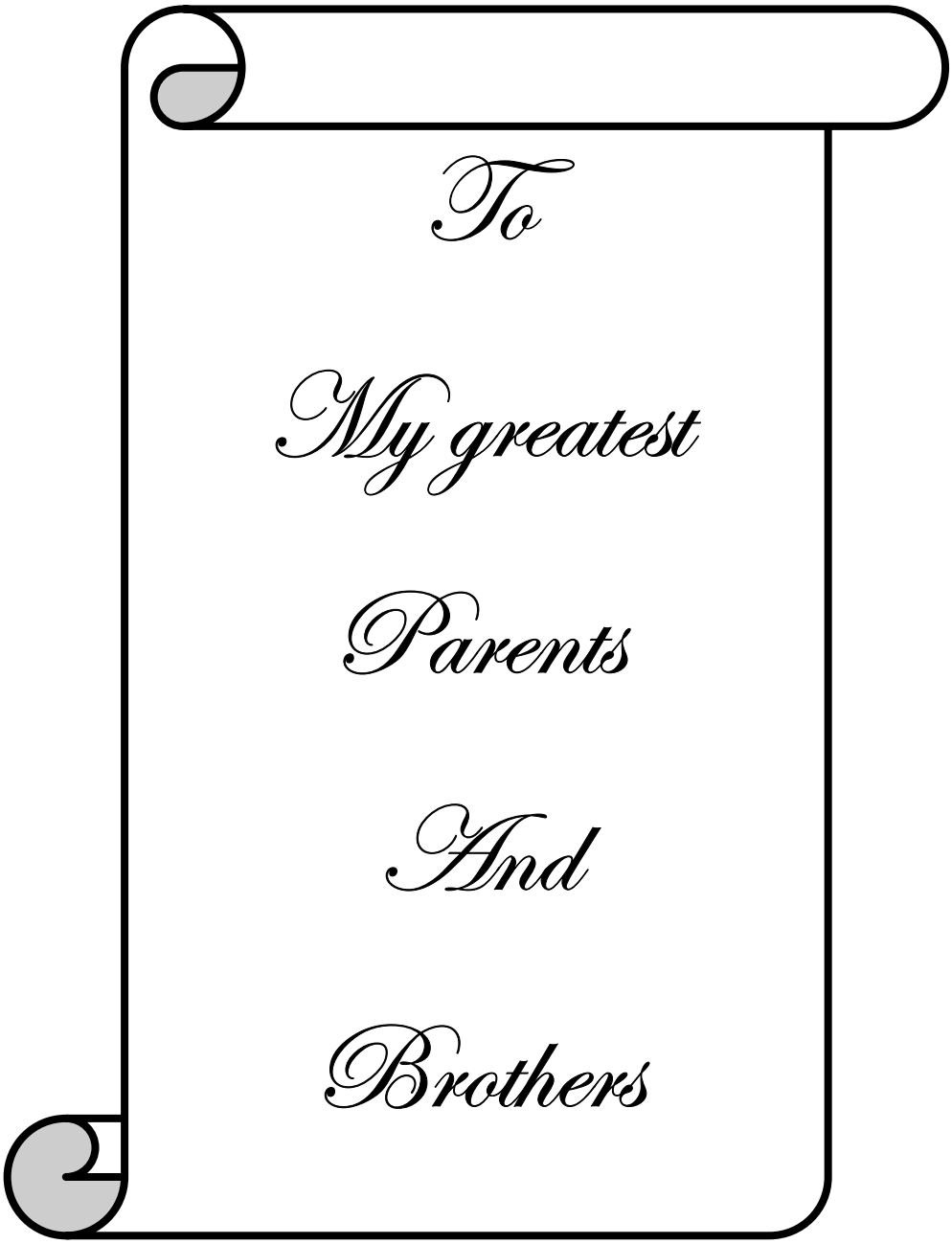
Dedication



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List of abbreviations

ABL	Alveolar bone loss
ALP	Alkaline phosphatase
BD	Bone density
BSP	Bone sialoprotein
CAL	Clinical attachment loss
ECM	Extracellular matrix
Eta_1	Early T-cell activation gene -1
FMLP	Formly –peptide
GAP	Generalized aggressive periodontitis
GCF	Gingival crevicular fluid
ICTP	Pyridinoline cross linked carboxy terminal telepeptide of type 1 collagen
IL	Interleukin
INFγ	Interferon gamma
INOS	Inducible nitric oxide synthase
LAP	Localized aggressive periodontitis
LPS	lipopolysacchrides
MMP	Matrix metalloproteinase
mRNA	Messenger Ribonucleic acid
NK	Natural killer
NO	Nitric oxide
OCN	Osteocalcin
ON	Osteonectin
OPG	Osteoprotegrin
OPN	Osteopontin
PBMCS	Peripheral blood mononuclear cells
PDGF	platelets derived growth factor
PGE2	Prostaglandin E2
PL	Periodontal ligament
PPD	Probing pocket depth
RGD	Argnine-glycine-aspartic acid (one letter code for Arg-Gly-Asp)
SIBLING	Small integrin –binding ligand N-linked glycoproteins
SVVYGLYR	Serine-Valine –valine –tyrosin-glycine-leucine-arginine
TGFβ	Transforming_ growth factor_ beta
TNFα	Tumor necrosis factor – alpha
TRAP	tartrate-resistant acid phosphatase

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Introduction

Periodontal disease which is marked by inflammation may result in subsequent loss and / or damage to tooth, supporting tissue including bone, cementum and periodontal ligament***. (polson and caton 1982)***. It is widely accepted that periodontal pathogens are accused for the initiation and sustenance of the inflammatory process in periodontal disease which is crucial for the destruction of mineralized and non-mineralized extracellular matrices in periodontal tissues ***(page 1991).*** Bacterial plaque products induce the differentiation of bone progenitor cells into osteoclasts and several host factors released by inflammatory cells are capable of inducing bone resorption in vitro and can play a role in periodontal disease ***(Schwartz et al., 1997).***These mediators are interleukin – 1, (1L-1) tumor necrosis factor – alpha, IL – 6 and prostaglandinE2 (PGE2**) (*Munday 1991*)** and some that are involved in inhibiting bone resorption like osteoprotegrin (***Mc Cauley and Nohuch 2002).***

The national institute of dental and craniofacial research performed concentrated research in salivary diagnostics, significant advancements have been achieved within the past 10 years using saliva, gingival crevicular fluid (GCF) and mucosal transudate as biological samples for the detection of oral and systemic illnesses (***Streckfus and Bigler 2002***). Salivary secretions are easily collected and containing local and systemic – derived biomarkers of periodontal disease (***Mandel 1993***).

Biomarkers of bone resorption or turnover include several biomarkers such as alkaline phosphatase (ALP) which is a catalyzing enzyme and its