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CHEMOTHERAPEUTIC APPROACH IN TREATMENT OF PERIODONTITIS IN EGYPTIAN POPULATION

A THESIS

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By

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قَالُوا سُبْحَانَكَ لا عِلْمَ لَنَا إلا مَا عَلَمْتَنَا إِلَّا مَا عَلَمْتَنَا إِلَّا مَا عَلَمْتَنَا إِلَّا مَا عَلَمْتَنَا إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ



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CONTENTS

CONTENTS

	Page
INTRODUCTION AND AIM OF THE WORK	1
LITERATURE REVIEW	4
Periodontal anatomy	4
Gingival crevicular fluid	8
Periodontal disease	12
Aggressive periodontitis	44
Nonsurgical periodontal therapy	51
 Control of periodontal disease for maintaining 	
general health	99
MATERIALS AND METHODS	103
Inclusion criteria	103
Exclusion criteria	103
The pilot study	104
Long-term study	105
 Assessment of response to pharmacotherapy 	107
Clinical assessments	107
Microbial assessments	109
Biochemical analysis	109
Drugs and chemicals	111
Statistical analysis	112
RESULTS	113
Prevalence of isolated pathogens	113
MIC of amoxicillin/clavulanate	115
MIC of doxycycline	115
MIC of metronidazole	115
MIC of ciprofloxacin	116
Clinical assessments	119
Microbial assessments	125
Biochemical analysis	136
DISCUSSION	146

LIST OF TABLES

	Page
Table (1): Mechanisms of reactive oxygen species (ROS) production in vivo.	27
Table (2): Prevalence of isolated pathogens from deepest pocket in pilot study of patients diagnosed as aggressive periodontitis.	114
Table (3): The mean minimal inhibitory concentrations (MIC) of metronidazole, amoxicillin/clavulanate, doxycycline and ciprofloxacin for isolated pathogens from deepest pocket in pilot study of patients diagnosed as aggressive periodontitis.	117
Table (4): Baseline and post-treatment results of clinical parameter groups of the long-term study.	120
Table (5): Prevalence of isolated pathogens from deepest	
pocket in long-term study of patients diagnosed as aggressive periodontitis at baseline and post-treatment.	129
Table (6): Baseline and post-treatment results of biochemical parameters in gingival crevicular fluid of patients in the long-term study.	139
Table (7): Number and percentage of adverse effects after medical treatment of aggressive periodontitis.	145

LIST OF FIGURES

	Page
Figure (1): The periodontium in health.	5
Figure (2): Established gingivitis lesion.	5
Figure (3): Periodontitis lesion	5
Figure (4): Peroxidation of unsaturated lipids.	29
Figure (5): Defense mechanism against damage by reactive oxygen species.	30
Figure (6): prevalence of isolated pathogens from deepest pocket in pilot study of patients diagnosed as aggressive periodontitis.	114
Figure (7): The mean minimal inhibitory concentrations of metronidazole, amoxicillin/clavulanate, doxycycline and ciprofloxacin for isolated pathogens from deepest pocket in pilot study of patients diagnosed as aggressive periodontitis.	118
Figure (8): Baseline and post-treatment results of gingival index in the five groups of the long-term study.	121
Figure (9): Baseline and post-treatment results of papillary	
Figure (10): Baseline and post-treatment results of probing	122
Figure (11): Baseline and post-treatment results of attachment	123
Figure (12): Prevalence of Actinobacillus actinomycetemcomitans from deepest pocket in long-term study of patients	124
diagnosed as aggressive periodontitis at baseline and post- treatment.	130
Figure (13): Prevalence of <i>Porphyromonas gingivalis</i> from deepest pocket in long-term study of patients diagnosed as	
aggressive periodontitis at baseline and post-treatment.	131
Figure (14): Prevalence of <i>Prevotella intermedia</i> from deepest pocket in long-term study of patients diagnosed as aggressive	

LIST OF ABBREVIATIONS

Actinobacillus actinomycetemcomitans	Aa
Aggressive periodontitis	AgP
Attachment level	ΑĽ
Black-pigmented bacteriods	BPB
Campylobacter rectus	Cr
Capnocytophaga sp	
Eikenella corrodens	
Gingival crevicular fluid	GCF
Gingival Index	GI
Minimal inhibitory concentration	MIC
Papillary bleeding index	PBI
Porphyromonas gingivalis	
Prevotella intermedia	
Probing Depth	PD
Reactive oxygen species	ROS
Standard error	S.E.
Thiobarbituric acid reactive substances	TBARS
Total antioxidant	TAO

DEDICATION

TO MY PARENTS, WIFE AND DAUGHTERS

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INTRODUCTION AND AIM OF THE WORK

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The term "periodontal disease" refers to both gingivitis and periodontitis. Gingivitis is an inflammatory condition of the soft tissues surrounding the teeth (the gingiva) and is a direct immune response to the dental microbial plaque building up on teeth. Periodontitis follows gingivitis and is also influenced by the individual's immune and inflammatory response. It is initiated by microbial plaque, but it occurs in only a subset of the population. Periodontitis involves the destruction of the supporting structures of the teeth including the periodontal ligament, bone and soft tissues (Kinane, 2001).

Aggressive periodontitis (AgP) generally affects systemically healthy individuals less than 30 years old, although patients may be older. Aggressive periodontitis may be universally distinguished from chronic periodontitis by the age of onset, the rapid rate of disease progression, the nature and composition of the associated subgingival microflora, alterations in the host's immune response, and a familial aggregation of diseased individuals (Loe and Brown, 1991)

According to American Academy of Periodontology, aggressive periodontitis was considered as high destructive periodontal diseases (AAP, 1999). Destructive periodontal diseases affect 10–15% of the world population and are a major cause of tooth loss (Brown and Loe, 1993). There is increasing evidence that the disease occurs in a predisposed group of the population that has an aberrant inflammatory/immune response to the microbial plaque that accumulates

around the gingival margin. This exaggerated response is known to result in inadvertent or collateral host tissue damage (Fredriksson et al., 1998).

Gingival crevicular fluid (GCF) is formed when fluid exudes from the vessels of the microcirculation into periodontal tissue and into the sulcus or pocket. As the fluid traverses the inflamed tissue, it is thought to pick up enzymes and other molecules that participate in the destructive process, as well as products of cell and tissue degradation (Perry et al., 1996). The fluid, therefore, offers great potential as a source of factors that may be associated with active tissue destruction. Efforts to develop diagnostic tests based on host factors have been focused almost entirely on analysis of GCF as malondialdehyde (degradation of lipids), total antioxidants (imbalance between free radicals and antioxidant) or iron (Mukherjec, 1985; Janero, 1990 Page, 1992; Chapple et al., 2002).

Collagen is the primary structural protein of all the periodontal tissues (comprising, for example, about 60% of total protein in gingiva and over 90% of the organic matrix of alveolar bone), and the destruction and loss of these fibers is the key event in the pathogenesis of the periodontal pocket (Narayanan and Page, 1983). A specific metalloprotease, collagenase, produced by several types of host cells which reside in or infiltrate the periodontal tissues (e.g., fibroblasts, polymorphonuclear leukocytes, macrophages) is largely responsible for this (Golub et al., 1976; Woolley and Davies, 1981). Also free radicals from host cells or microorganisms were responsible for the destruction of these structures leading to produce a specific compound was called malondialdehyde (Esterbauer and Cheeseman, 1990; Bax et al., 1992; Hall et al., 1995)

Gingival crevicular fluid (GCF) was analyzed to assess the inflammatory process in biochemical terms, into three general groups: 1)