بسم الله الرحمن الرحيم

شبكة المعلومـــــات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

﴿ قـــســـم ﴾

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأفلام قد اعدت دون آية تغيرات

يجب أن

تحفظ هذه الأفلام بعيداً عن الغبار

في درجة حرارة من 15 – 20 مئوية ورطوبة نسبية من 20-40 %

To be kept away from dust in dry cool place of
15 – 25c and relative humidity 20-40 %

بعض الوثائق الأصلية تالفة

بالرسالة صفحات

لم ترد بالأصـــل

EFFECT OF SELECTIVE BETA-2 AGONIST ON THE GINGIVA OF ASTHMATIC PATIENTS CLINICAL, HISTOLOGICAL AND HISTOCHEMICAL STUDY

A Thesis Submitted
In Partial Fulfillment of the
Requirements of the

MASTER DEGREE

In

Oral Medicine, Periodontology,
Oral Diagnosis and
Oral Radiology

By

MANAL NAIM DOWIDAR

Faculty of Dentistry
Alexandria University
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SUPERVISORS

Prof. Dr. Eglal M. Moussa

Professor of Oral Medicine,
Periodontology, Oral Diagnosis and Oral Radiology
Faculty of Dentistry
Alexandria University

Prof. Dr. Nasrah A. Shaaban

Professor of Histology
Faculty of Medicine
Alexandria University

Dr. Aly M. Sadek

Lecturer of Oral Medicine,
Periodontology, Oral Diagnosis and Oral Radiology
Faculty of Dentistry
Alexandria University

Prof. Dr. Mohammed Ehab M. Atta

Professor of Chest Diseases
Department of Chest Diseases
Faculty of Medicine
Alexandria University

To My
Parents,
My Husband,
And My Daughters
Radwa & Salma

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CHAPTER 1

Introduction

INTRODUCTION

Asthma is one of the few chronic diseases in the world that is increasing in prevalence despite better understanding of its pathogenesis and improved treatment. The past decade has seen a major shift in the perception of asthma. It has gone from being considered a disease primarily characterized by altered smooth muscle response to one whose main abnormality is chronic inflammation.[1]

Recently, asthma is defined as *"a chronic inflammatory disorder of the airways in which many infiltrating cells play a role"*, in particular mast cells which are followed by eosinophils, and T-lymphocytes that are called to the site of inflammation by the chemotactic products produced by activated mast cells.[2-4] Upon their arrival, these cells release their own products of inflammation which amplify their immunologic response.[5]

In susceptible persons, this inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness and cough particularly at night and in the early morning.[2,6]

These symptoms are usually associated with wide spread but variable airflow limitation that is at least partly reversible, either spontaneously or with treatment.[2,7]

Mast cells are one of the inflammatory cells that play a pivotal role in the pathogenesis of asthma.[8] Its main function has centered on the pathologic role of inflammatory mediators in extruded mast cell granules.[9]

The relative role of mast cells depends on the immunologic sensitivity of the host, the target tissue involved and any underlying pathology.[10]

The main etiological factor in extrinsic atopic asthma is the immediate allergic hypersensitivity which results from the interaction of an antigen and antibodies (*immunoglobulin*) which belongs mainly to the E-class.[11] The IgE antibodies have a special affinity for the mast cells and basophilic leukocytes and attach themselves to the surface of these cells. The interaction between the antigen and antibodies leads to degranulation of mast cells, dissolution of its free granules,[12] that release several mediators like histamine and neutrophil chemotactic factor and induces release of membrane derived mediators as leukotriens and prostaglandins from the membrane of mast cells.[13,14] These mediators activate several target cells in the airway, resulting in bronchoconstriction, microvascular leakage with oedema as well as mucous hypersecretion and stimulation of neural reflexes.[15]

There are several groups of drugs in use to improve the ventilatory status in asthmatics. Some are used to treat acute attacks, while others work better for the prevention of future episodes. Each acts at a certain level of the pathologic process of asthma.[16] Therapy in acute attacks should always include an adrenergic agent, such as B_2 adrenergic agonists which act mainly as bronchodilator and are also effective mast cell stabilizer[17] and they can reduce plasma leak and exudation from the bronchial microcirculation.[18]

Salbutamol is one of the selective B_2 agonists that is considered by some authors as the potent and safest sympathomimetic bronchodilator by its relaxant effect on the bronchial smooth muscle and by enhancing mucociliary clearance.[19] It also has an anti-allergic effect as it inhibits the release of mast cells mediators.[20,21]

Previous studies have shown that children suffering from asthma had more gingivitis than their healthy controls without difference in the amount of dental plaque.[22] Also, it is known that the number of mast cells changes in gingivitis.[23] Some authors had proposed an inverse relationship between mast cell density and the degree of inflammation.[24-26] While others found that the number of mast cells increases in presence of chronic gingivitis than it is in the normal gingiva.[27-29] So, because mast cells have got a pivotal role in asthma and in the same time, they play a role in gingival inflammation, so further research using histochemical technique specific for the amount of mast cells in the gingiva of asthmatic patients was recommended.

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CHAPTER II

Review Of Literature