FUNCTIONAL STATE OF THE CALCIUM-REGULATING SYSTEM AND ITS RELATION TO GROWTH RETARDATION IN PATIENTS WITH BRONCHIAL ASTHMA

Thesis

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بسم الله الرحمن الرحيم

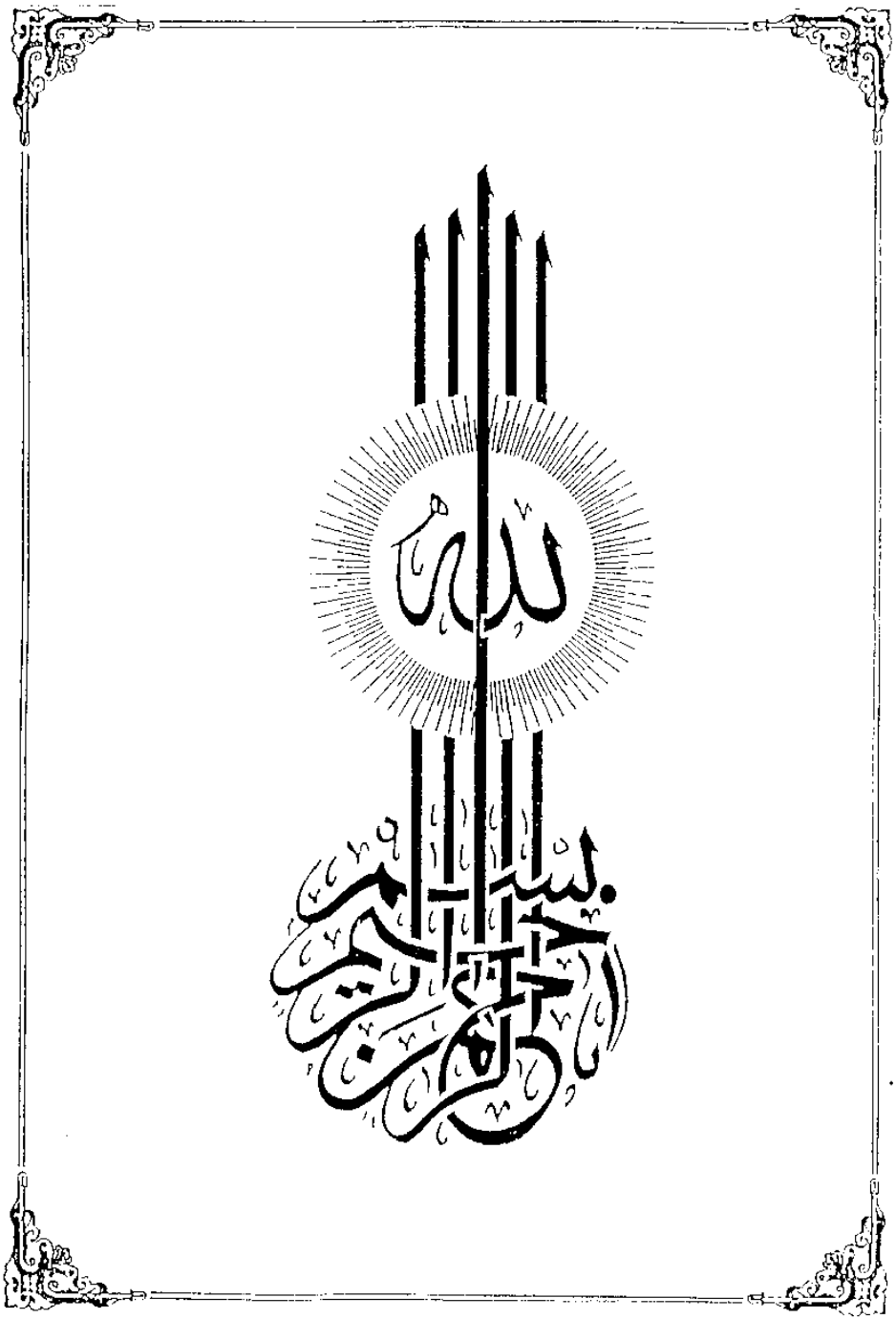
اقرأ باسم ربك الذي خلق

خلق الإنسان من علق

اقرأ وربك الأكرم الذي علم بالقلم

علم الإنسان ما لم يعلم

العلق ١-٥



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LIST OF ABBREVIATIONS

- **BMD** : Bone maturity difference.
- **cAMP** : Cyclic adenosine monophosphate.
- **CCK8** : Cholycystokinin 8.
- **CGRP** : Calcitonin gene-related peptide.
- **ECP** : Eosinophil cationic protein.
- **EPO** : Eosinophil peroxidase.
- **FEV** : Forced expiratory volume.
- **ICAM-1** : Intercellular adhesion molecule-1.
- **IL** : Interleukin.
- **LT** : Leukotriene.
- **LFA-1** : Lymphocyte function-associated antigen-1.
- **MBP** : Major basic protein.
- **PAF** : Platelet-activating factor.
- **PEFR** : Peak expiratory flow rate.
- **PG** : Prostaglandin.
- **PTH** : Parathyroid hormone.
- **RSV** : Respiratory syncytial virus.
- **TNF** : Tumor necrosis factor.
- **VIP** : Vasoactive intestinal peptide.

LIST OF TABLES

Table (13):
Sex distribution in asthmatic groups.

Table (14):
Mean weight percentile in asthmatic groups.

Table (15):
Mean height percentile in asthmatic groups.

Table(16):
Asthmatics below 10th percentiles for weight and height.

Table (17):
Relation between severity of asthma and weight percentile.

Table (18):
Relation between hospital admission and weight percentile.

Table (19):
Mean bone age in asthmatic groups.

Table (20):
Mean BMD in asthmatic groups.

Table (21):
Relation between BMD and duration of asthma.

Table (22):
Mean chronological age, bone age and BMD in cases and control groups.

Table (23):
Calcium level in cases and controls.

LIST OF FIGURES

CONTENTS

INTRODUCTION AND AIM OF THE WORK

INTRODUCTION

Asthma is a leading cause of chronic illness in childhood. It is responsible for a significant proportion of school days lost because of chronic illness. It is also the most frequent admitting cause in children's hospital. It was estimated that 5-10% of children sometime during their childhood had signs and symptoms compatible with asthma.

Asthmatics children may have growth retardation unrelated to corticosteriod administration (Ellis, 1983).

Growth retardation occurs in all parameters with varying degrees in asthmatic children (Abdel Khalek et al., 1986).

Marked retardation of skeletal maturation as a general phenomenon of atopic asthma was described by Baum et al. (1990).

Disturbance in calcium regulation system appear in the course of the disease progression and is augmented in long term glucocorticoid treatment (Chuchalin and Berova, 1989).

AIM OF THE WORK:

* To find out relation between bronchial asthma, its severity and duration and retarded physical growth.

* To show whether this physical retardation is related to changes in calcium homeostasis or not.

REVIEW OF LITERATURE

BRONCHIAL ASTHMA

Introduction:

Asthma is an inflammatory disease of the airway that is chronic and persistent (Lee, 1992).

Asthma is defined by the American Thoracic Society in 1987 as the presence of intermittent symptoms that include wheezing, dyspnea and cough resulting from airway hyperreactivity and reversible airflow obstruction. Asthma is often associated with atopy, like other inflammatory diseases, asthma is characterized by the recruitment of inflammatory cells, vascular congestion, increased vascular permeability, increased tissue volume and the presence of an exudate (Lee, 1992).

Asthma is characterized by more or less pronounced hyperreactivity of bronchial tissue to physical, pharmacological and/or immunological stimuli (Aas et al., 1981).

The bronchial obstruction may be brought about by spasm of bronchial muscles, mucus secreted into the bronchial lumen, oedema of the mucosa or by combination of the three factors (Simpson, 1980).

Asthmatic inflammation in an atopic subject may be distinguished from other inflammatory diseases by a characteristic pattern of early mast cell activation, eosinophil infiltration, fibroblast proliferation and collagen deposition, selective T-cell activation, epithelial damage and mucus hypersecretion (Gleich et al., 1988).

Aetiology of Asthma:

Asthma is a complex disorder involving autonomic, immunologic, infectious, endocrine and psychological factors in varying degrees in different individuals (Huchanf and Madia, 1987).

1) Autonomic factors:

The walls of bronchi and bronchioles are innervated by the autonomic nervous system. There are abundant muscarinic receptors, and cholinergic discharge that causes bronchoconstriction. There are B1 and B2 adrenergic receptors in the bronchial epithelium and smooth muscle and in mast cells. Many are not innervated. However, some may be located on cholinergic endings and ganglia, where they inhibit acetylcholine release. In humans, the B2 receptors predominate and inhaled or injected B agonists such as isoproterenol cause bronchodilatation and decreased bronchial secretion. Some have argued that there is an imbalance between the muscarinic receptors mediating bronchoconstriction and the B adrenergic receptors mediating bronchodilatation and the disease generally responds well to inhaled B2 adrenergic agonists (Insel and Wasserman, 1990).

The neural reaction is believed to be in sensory receptors in large airways. These receptors in asthmatic patients are hyperactive and their stimulation by antigenic and non antigenic stimuli, leads to vagal reflex that results in reflex bronchoconstriction (Ellis, 1983).

One of the absolute features of asthma is an exaggerated airway reactivity to irritating