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التوثيق الالكتروني والميكروفيلم

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شبكة المعلومات الجامعية

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التوثيق الإلكتروني والميكروفيلم

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لم ترد بالأصل

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قال رب اشرح لى صدرى
ويسر لى أمرى واحلل عقدة
من لسانى يفقهوا قولى

سورة طه (الأيات ٢٥ إلي ٢٨)

الرسالة طابقة للمواصفات

SERUM LEVEL OF SOLUBLE INTERLEUKIN-2 RECEPTOR DURING ACUTE ASTHMA EXACERBATION AND ITS RELATION TO SEVERITY AND RESPONSE TO BRONCHODILATOR THERAPY

تمت مناقشة الرسالة مناقشة علنية في مستشفى الأطفال الجامعي بسموحة الأطفال
يوم ٢٢/١٠/٢٠٠١ وقبلت بتقدير ممتاز ١٠٠٪

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By

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To My

Parents and Brother

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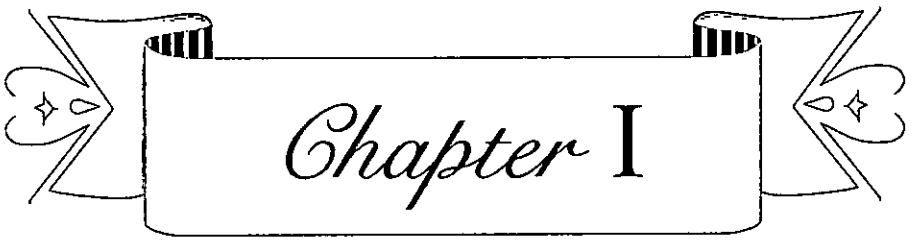

INTRODUCTION

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Acute bronchial asthma is a common problem with immence medical and economic impacts.[1] Most of morbidity and all of the mortality of asthma tend to be associated with acute exacerbations, and treatment of these events accounts for the majority of expenditures of money and health care resources. However, the factors that contribute to the destabilization of the disease are little studied and much of the pathogenesis and pathobiology of acute asthma remains unkown.[2]

Asthma may have its onset at any age; 30% of patients are symptomatic by one year of age, whereas 80-90% of asthmatic children have their first symptoms before 4-5 years of age. As many as 10-15% of boys and 7-10% of girls may have asthma at sometime during childhood. Before puberty, approximately twice as many boys as girls are affected; thereafter, the sex incidence is equal.[3] In Egypt, asthma is quite common and affects approximately 8.2% of children in the age groups 3-15 years.[4]

Both prevalence and mortality from asthma have increased during the last three decades.[5] The causes of increased prevalence are unknown, but some of the factors associated with the onset of asthma and increased

mortality have been identified.[6] Risk factors of occurrence of asthma include poverty, maternal age less than 20 years at time of birth, birth weight less than 2500 g, maternal smoking, small home size, large family and intense allergic exposure in infancy.[7] Additional risk factors, may include frequent respiratory infections in early childhood.[6] Risk factors for death from asthma include underestimation of severity of asthma, delay in implementation of appropriate treatment, underuse of bronchodilators and corticosteroids, non compliance with recommendations for management, psychological dysfunction and stress that may interfere with compliance or perceptions of increasing airway obstructions, sedation, and excessive allergic exposure.[8]

There is no universally accepted definition of bronchial asthma. However, it may be considered as a complex disorder of diffuse bronchial inflammation and smooth muscle hyperreactivity leading to obstructive lung disease in response to various stimuli. The obstructed airways are characterized by a high degree of reversibility of the obstructive forces which may occur spontaneously or in response to treatment.[3,9]

The exact mechanism by which acute episodes of asthma are initiated and sustained is still unknown.[10,11] This disorder in asthma may involve autonomic, immunologic, infectious, endocrinal, and psychological factors

in varying degrees in different individuals.[12] The common pathway by which all these factors whether immunologic or non immunologic increase airway resistance is inflammation.[13] Airway inflammation in asthma contributes to long term bronchial hyper-responsiveness, airflow limitation and airway remodeling.[14] The increase in airway resistance results in decreased forced expiratory volume and flow rates, premature airway closure, hyperinflation of the lungs and hemithoraces, increased work of breathing, changes in elastic recoil, and frequently dependent behaviour of the lung.[15,16,17,18] In addition, there is abnormal distribution of both ventilation and perfusion and altered arterial blood gas levels.[2,16,19] In very symptomatic patients, there can be electrocardiographic evidence of pulmonary hypertension, right ventricular strain and compromised left ventricular filling.[2,15,20]

It is apparent that asthma is not caused by either single cell or a single inflammatory mediator but rather results from complex interactions among inflammatory cells, mediators and other tissues resident in airways.[21]

Pulmonary response in asthma includes early-phase and late phase reactions. Early-phase reactions occur within 10-30 minutes and resolve within 2 hours. Late-phase reactions usually occur within 4-8 hours and may last for 24 hours or longer. Early reactions after antigenic[3,21] stimulation include vasodilation, edema formation from increased vascular permeability,