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قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات

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بالرسالة صفحات لم ترد بالأصل

SERUM LEVEL OF SOLUBLE INTERCELLULAR ADHESION MOLECULE-1 ($_{s}$ICAM -1) IN ACUTE ATTACKS OF BRONCHIAL ASTHMA IN CHILDREN.

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To My Family and My Friends

بسم الله الرحمن الرحيم

ABBREVIATIONS

CD	Clusters of differentiation
E-selectin	Endothelial-selectin
ICAM-1	Intercellular adhesion molecule – 1
$_{s}$ICAM-1	Soluble intercellular adhesion molecule –1
IL	Interleukin
LFA –1	Lymphocyte (leukocyte) function associated antigen –1
L-selectin	Leukocyte-selectin
Mac –1	Macrophage antigen – 1
MAdCAM-1	Mucosal addressin cellular adhesion molecule –1
PECAM –1	Platelet-endothelial cell adhesion molecule-1
PML	Polymorphonuclear Leukocytes
P-selectin	Platelet – selectin
TNF	Tumour necrosis factor
VCAM –1	Vascular cell adhesion molecule –1
VLA- 4	Very late activation antigen – 4

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Chapter I

INTRODUCTION

INTRODUCTION

Bronchial asthma is a leading cause of chronic illness in childhood, responsible for a significant proportion of school days lost because of chronic illness [1]. Asthma is the most frequent admitting diagnosis in children's hospitals and there has been an increase in hospitalization rates for asthma in children worldwide [2]. As many as 10 – 15% of boys and 7 –10 % of girls may have asthma at some time during childhood . Before puberty approximately twice as many boys as girls are affected; thereafter, the sex incidence is equal [1]. In Egypt, asthma is quite common and affects approximately 8.2% of children aged three to fourteen years [3].

The increasing morbidity [3] and fatality [4] from asthma have been partly explained by several factors including the change in the International Classification of Disease version 8 and 9 which resulted in "asthmatic bronchitis" being coded as asthma rather than bronchitis, actual increases in prevalence or severity of asthma [5,6], identification of sociologic and biologic risk factors [5,6,7], possible effects of drug toxicity [8,9] and geographic or environmental effects [10,11].

There is no universally accepted definition of asthma; it may be regarded as a diffuse, obstructive lung disease with hyperreactivity of the

airways to a variety of stimuli and a high degree of reversibility of the obstructive process, which may occur either spontaneously or as a result of treatment [1]. In addition to bronchoconstriction, inflammation is an important pathophysiologic factor; it involves eosinophils, monocytes and immune mediators and has resulted in the alternative designation of chronic desquamating eosinophilic bronchitis [1].

Pathogenesis of bronchial asthma:

Asthma is a complex disorder involving autonomic, immunologic, infectious, endocrine, and psychologic factors in varying degrees in different individuals [1]. It is evident that quite a large number of factors interplay in the pathogenesis of asthma. The common pathway by which all these factors, whether immunological or non-immunological, increase airway resistance, is inflammation. [12]

Mucosal inflammation is an integral component of the asthmatic airways and is considered to be central in the pathogenesis of airway – dysfunction [13,14]. The bronchial mucosa of asthmatic patients is characterized by a large influx of eosinophils, monocytes and lymphocytes [15]. Accumulation of eosinophil granulocytes is a common finding in lung tissue of patients with asthma, especially during an active state of the disease [16,17]. The accumulated eosinophils are considered to play an important role in the