Socioeconomic and Personal Correlates of Bronchial Asthma in Suez Chest Hospital

Thesis

Submitted for partial fulfillment of the Master degree in
Chest diseases & Tuberculosis

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2013

Acknowledgment

In the name of Allah, most gracious, most merciful.

First of all I would like to thank Allah who enabled me to finish this research.

I am deeply indebted to my supervisor; Professor **Emad Eldine AbdelWahab Korraa**; Professor of Chest diseases, Ain Shams University whose guidance and instructions were invaluable.

I would like to express my gratitude to Doctor **Hala Mohammad Salem**; Assistant professor of Chest Diseases, Ain Shams University for her help & patience all through the time of the research.

I forward my appreciation to all the staff of Suez Chest Hospital; doctors and nurses for their tremendous efforts to help me to finish this research.

Finally, I would like to thank all my family for their support, help and love.

Introduction

Asthma is a chronic inflammatory disorder of the airways that causes recurrent episodes of airflow limitation characterized by wheezing, breathlessness, chest tightness, and cough in susceptible individuals, and is spontaneously reversible or reversible on treatment.[(1)]

The ***World Health Organization (WHO)*** estimates that 300 million people currently suffer from asthma, resulting in a global loss of approximately 15 million disability-adjusted life years (DALYs) annually.[(2)] Although exact epidemiological data are lacking, its prevalence is estimated to be approximately 5.5% of the general population. Most people develop it before the age of 30 years .[(3)] In Cairo, Egypt, the prevalence of bronchial asthma was reported to be 9.4%.[(4)]

Socioeconomic status may be particularly relevant to asthma due to pathways by which it could adversely impact asthma outcomes. At the individual level (e.g., education attainment, income), asthmatics of lower socioeconomic status may have higher exposures to indoor (e.g., cockroaches, tobacco smoke) and outdoor (e.g., urban pollution) allergens, and tend to use less inhaled corticosteroids,[(5)] thus increasing risk for acute asthma exacerbations .Though the socioeconomic status asthma link has been well established in children and to some degree in adults, less is known about associations between individual-level socioeconomic and asthma in adults.[(6)]

Numerous epidemiological studies have investigated the associations of risk factors with subjective (i.e. questionnaire reported asthma or wheeze) and objective (i.e. lung function measures and bronchial provocation challenges) adult measures of respiratory health. Several interrelated factors, including

genetics,[7] childhood exposures and respiratory symptoms,[8] allergic sensitization, exposure to cigarette smoke, obesity, sex hormones, indoor environmental factors, outdoor air pollution, and certain occupational exposures have been identified as risk factors for adult asthma, with consistent associations found in multiple studies and settings.[9]

The asthma control test survey, a clinically validated measure of asthma control that is simple to administer, should be useful to clinicians who are interested in assessing asthma control in patients within their practice and to investigators seeking to assess the level of asthma control within a population, with or without the use of lung function testing.[10]

The asthma control test is an easily administered and scored survey that measures asthma control accurately compared with specialist ratings on the basis of history, physical examination, and lung function tests and with the previously validated asthma control test. The asthma control test survey should facilitate efforts to improve assessment of asthma control in the busy clinical practice setting.[10]

Aim of the work

The aim of this work is to study the socioeconomic and personal characteristics of bronchial asthma patients and its relation to asthma control in Suez chest hospital.

Bronchial Asthma

Definition

Asthma is a chronic inflammatory disorder of the air ways in which many cells and cellular elements play a role. The chronic inflammation is associated with airway hyper responsiveness that leads to recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night, or in the early morning. These episodes are usually associated with wide spread, but variable, airflow obstruction within the lung that is often reversible either spontaneously or with treatment.[(11)]

Epidemiology

Asthma affects an estimated 300 million individuals worldwide. The overall prevalence rate of exercise-induced bronchospasm is 3-10% of the general population if persons who do not have asthma or allergy are excluded, but the rate increases to 12-15% of the general population if patients with underlying asthma are included. Annually, the ***World Health Organization (WHO)*** has estimated that 15 million disability-adjusted life-years are lost and 250,000 asthma deaths are reported worldwide.[(12)]

There is evidence that its prevalence has increased considerably over the past 20 years, especially in children. ***The Global Initiative for Asthma (GINA),2011***[(11)] was created to increase awareness of asthma among health professionals, public health authorities, and the general public, and to improve prevention and management through a concerted worldwide effort.[(13)]

Asthma predominantly occurs in boys in childhood, with a male-to-female ratio of 2:1 until puberty, when the male-to-female ratio becomes 1:1. Asthma prevalence is greater in females after puberty, and the majority of adult-onset cases diagnosed in persons older than 40 years occur in females. Boys are more likely than girls to experience a decrease in symptoms by late adolescence. Asthma prevalence is increased in very young persons and very old persons because of airway responsiveness and lower levels of lung function.[14]

The rate of asthma increases as communities adopt western lifestyles and become urbanized. With the projected increase in the proportion of the world's population that is urban from 45% to 59% in 2025, there is likely to be a marked increase in the number of asthmatics worldwide over the next two decades. It is estimated that there may be an additional 100 million persons with asthma by 2025.[15]

Pathophysiology

The pathophysiology of asthma is complex and involves airway inflammation, intermittent airflow obstruction and bronchial hyperresponsiveness. The mechanism of inflammation in asthma may be acute, subacute, or chronic. Some of the principal cells identified in airway inflammation include mast cells, eosinophils, epithelial cells, macrophages, and activated T lymphocytes. Other constituent airway cells, such as fibroblasts, endothelial cells, and epithelial cells, contribute to the chronicity of the disease.[16]

Airflow obstruction can be caused by a variety of changes, including acute bronchoconstriction, airway edema, chronic mucous plug formation, and airway remodeling. Acute bronchoconstriction is the consequence of immunoglobulin E-dependent mediator release upon exposure to aeroallergens and

is the primary component of the early asthmatic response. Airway edema occurs 6-24 hours following an allergen challenge and is referred to as the late asthmatic response. Chronic mucous plug formation consists of an exudate of serum proteins and cell debris that may take weeks to resolve. Airway remodeling is associated with structural changes due to long-standing inflammation and may profoundly affect the extent of reversibility of airway obstruction.*(17)*

Hyperinflation compensates for the airflow obstruction, but this compensation is limited when the tidal volume approaches the volume of the pulmonary dead space; the result is alveolar hypoventilation. Uneven changes in airflow resistance, the resulting uneven distribution of air, and alterations in circulation from increased intra-alveolar pressure due to hyperinflation all lead to ventilation-perfusion mismatch. Vasoconstriction due to alveolar hypoxia also contributes to this mismatch. Vasoconstriction is also considered an adaptive response to ventilation/perfusion mismatch. *(18)*

In the early stages, when ventilation-perfusion mismatch results in hypoxia, hypercarbia is prevented by the ready diffusion of carbon dioxide across alveolar capillary membranes. Thus, patients with asthma who are in the early stages of an acute episode have hypoxemia in the absence of carbon dioxide retention. Hyperventilation triggered by the hypoxic drive also causes a decrease in $PaCO_2$. An increase in alveolar ventilation in the early stages of an acute exacerbation prevents hypercarbia. With worsening obstruction and increasing ventilation-perfusion mismatch, carbon dioxide retention occurs. In the early stages of an acute episode, respiratory alkalosis results from hyperventilation. Later, the increased work of breathing, increased oxygen consumption,

and increased cardiac output result in metabolic acidosis. Respiratory failure leads to respiratory acidosis. [18]

Recent studies have suggested that polymorphism of the beta-2 adrenergic receptor (b2AR) gene at codon 16 affect an individual's airway responsiveness.[19]

The common variant IL-13 gene polymorphism is reported to be associated with the risk of development of asthma in some populations. ***El-Sayed et al., 2010*** [20] sought to study the association of IL-13 genetic variant R130Q with bronchial asthma in Egyptian children and its relation to various clinical and laboratory phenotypes of the disease. IL13 gene polymorphism (R130Q) was detected by PCR in 20 asthmatic patients with acute exacerbation. The results were compared to 20 healthy age and sex matched children. They found that Asthmatic children had significantly higher frequency of distribution of R130Q genotype (50%) than controls (15%). The serum total IgE as percent of high normal for age was significantly higher in asthmatic patients as compared to controls.

Prognosis

International asthma mortality is reported as high as 0.86 deaths per 100,000 persons in some countries. US asthma mortality rates in 2006 were reported at 1.2 deaths per 100,000 persons. Mortality is primarily related to lung function, with an 8-fold increase in patients in the lowest quartile, but mortality has also been linked with asthma management failure, especially in young persons. Other factors that impact mortality include age older than 40 years, cigarette smoking more than 20-pack years, blood eosinophilia, and forced expiratory volume in one second (FEV_1) of 40-69% predicted.[18]

Diagnosis of bronchial asthma

General manifestations of asthma

Wheezing is one of the most common symptoms. In the mildest form, wheezing is only end expiratory. As severity increases, the wheeze lasts throughout expiration. In a more severe asthmatic episode, wheezing is also present during inspiration. During a most severe episode, wheezing may be absent because of the severe limitation of airflow associated with airway narrowing and respiratory muscle fatigue. *(21)*

Episodic symptoms after an incidental allergen exposure, seasonal variability of symptoms and a positive family history of asthma and atopic disease are also helpful diagnostic guides. Asthma associated with rhinitis may occur intermittently, with the patient being entirely asymptomatic between seasons or it may involve seasonal worsening of asthma symptoms on a background of persistent asthma. The patterns of these, symptoms that strongly suggest an asthma diagnosis are variability; precipitation by non-specific irritants, such as smoke, fumes, strong smells, or exercise: worsening at night; and responding to appropriate asthma therapy.*(11)*

Asthma can occur without wheezing when obstruction involves predominantly the small airways. Thus, wheezing is not necessary for the diagnosis of asthma. Furthermore, wheezing can be associated with other causes of airway obstruction, such as cystic fibrosis and heart failure. In exercise-induced asthma, wheezing may be present after exercise, and in nocturnal asthma, wheezing is present during the night. Cough may be the only symptom of asthma, especially in cases of exercise-induced or nocturnal asthma. Usually, the cough is nonproductive and nonparoxysmal. *(21)*

Physical Examination

The updated guidelines from the National Asthma Education and Prevention Program (NAEPP) highlight the importance of correctly diagnosing asthma, by establishing the following:[22]

- Episodic symptoms of airflow obstruction are present.
- Airflow obstruction or symptoms are at least partially reversible.
- Exclusion of alternative diagnoses.

Table (1): Classification of Asthma Severity by Clinical Features:[23]

Intermittent
Symptoms less than once a week. Brief exacerbations. Nocturnal symptoms not more than twice a month. FEV1 or PEF > 80% predicted. FEV1 or PEF variability < 20%.
Mild Persistent
Symptoms more than once a week but loss than once a day Exacerbations may affect activity and sleep Nocturnal symptoms more than twice a month • FEV1 or PEF > 80% predicted • FEV1 or PEF variability < 20-30%
Moderate Persistent
Symptoms daily. Exacerbations may affect activity and sleep. Nocturnal symptoms more than once a week. Daily use of inhaled short-acting B_2-agonist. FEV1 or PEF 60-80% predicted. PEF or FEV1 variability > 30%.

Severe Persistent
Symptoms daily. Frequent exacerbations. Frequent nocturnal asthma symptoms. Limitation of physical activities. FEV1 or PEF < 60% predicted. FEV1 or PEF variability >30%.

Severity of acute asthma[24]

Moderate

- Increasing symptoms
- PEFR 50-75% predicted best
- No features of acute severe asthma
- One hour following treatment in A&E, patients with PEF > 75% predicted or best, may be discharged home with appropriate changes to their asthma medication, in the absence of concerns, e.g.
    - Significant ongoing symptoms.
    - Compliance concerns.
    - Living alone.
    - Psychological problems or learning difficulties.
    - Previous near fatal or brittle asthma.
    - Nocturnal presentation.
    - Pregnant.
    - Exacerbation despite adequate oral steroid pre-presentation.

Severe asthma

Any one of:

- PEFR 33-50% predicted or best.
- RR > 25.
- HR > 110.
- Inability to complete sentence in one breath.

Life-threatening asthma

Any one of:

- PEFR < 33%.
- SaO_2 < 92 % (needs ABG).
- PaO_2 < 8 kPa(60 mmhg).
- Normal CO_2.
- Silent chest.
- Cyanosis.
- Poor respiratory effort.
- Bradycardia/ arrhythmia/ hypotension.
- Exhaustion.
- Confusion.
- Coma.

Near fatal asthma

- Raised $PaCO_2$ and/or
- Needing mechanical ventilation with raised inflation pressures.

N.B. Brittle asthma:Type 1: wide PEF variability (>40% diurnal variation for >50% of the time over >150 days), despite appropriate therapy. **Type 2**: sudden severe attacks on background of apparently good control.[(24)]

Non pulmonary Manifestations of bronchial asthma

Signs of atopy or allergic rhinitis, such as conjunctival congestion and inflammation, a transverse crease on the nose due to constant rubbing, and pale violaceous nasal mucosa due to allergic rhinitis, may be present in the absence of an acute episode, such as during an outpatient visit between acute episodes. Skin examination may reveal atopic dermatitis, eczema, or other manifestations of allergic skin conditions.[(18)]

Diagnostic Considerations

Vocal cord dysfunction

Vocal cord dysfunction may exist alone or with asthma, it is caused by paradoxical adduction of the vocal cords during inspiration, and may disappear with panting, speech, or laughing. Patients with chronic symptoms suggestive of asthma, normal spirometry, poor response to asthma medications, and frequent evaluations should be evaluated for vocal cord dysfunction.[(25)]

Patients with vocal cord dysfunction have a predominantly inspiratory monophonic wheeze (different from the polyphonic wheeze in asthma), which is heard best over the laryngeal area in the neck. Patients with bronchomalacia and tracheomalacia also have a monophonic wheeze. In exercise-induced asthma, wheezing may be present after exercise, and in nocturnal asthma, wheezing is present during the night.[(21)]

Cough-variant asthma

Some patients with asthma have chronic cough (frequently occurring at night) as their principal, if not only, symptom. For these patients, documentation of lung function variability and airway hyperresponsiveness are particularly important.[(13)]

Exercise-induced bronchoconstriction

In patients with exercise-induced bronchospasm, the clinical history findings are typical of asthma but are associated only with exercise. Typical symptoms include cough, wheezing, shortness of breath, and chest pain or tightness. Initially, airway dilation is noted during exercise. If exercise continues beyond approximately 10 minutes,

bronchoconstriction supervenes, resulting in asthma symptoms. If the exercise period is shorter, symptoms may develop up to 5-10 minutes after completion of exercise. A higher intensity level of exercise results in a more intense attack, with running producing more symptoms than walking.[20]

Children under age 5

Not all young children who wheeze have asthma. In this age group, the diagnosis of asthma must be based largely on clinical judgment, and should be periodically reviewed as the child grows.[13]

Foreign bodies

Foreign body aspiration may cause not only localized wheezing but also generalized wheezing.[26]

Asthma in the elderly

Diagnosis and treatment of asthma in the elderly are complicated by several factors, including poor perception of symptoms, acceptance of dyspnea as being "normal" for old age, and reduced expectations of mobility and activity. While diagnosing asthma it is crucial to exclude COPD, the simplest way to differentiate between these two diseases is symptoms.[13]

Pulmonary migraine

Pulmonary migraine consists of combined recurrent asthma; cough with thick mucoid sputum; lower back pain radiating to the shoulder; subtotal or total atelectasis of a segment or lobe; and, occasionally, nausea with vomiting. The symptoms are often accompanied closely in time by focal headache.[27]