Prevalence Of Asthma-Chronic Obstructive Pulmonary Disease Overlap Syndrome (ACOS) In Embaba Chest Hospital

Thesis

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In Chest Disease and Tuberculosis

By **Ahmed Abd Elhameed Bahieg**M.B.B.ch

Supervised by

Prof. Dr./Emad El Din Abdel Wahab Korraa

Professor of chest Diseases and Tuberculosis Faculty of Medicine – Ain Shams University

Dr. Rehab Maher

Lecturer of Chest Diseases and Tuberculosis Faculty of Medicine – Ain Shams University

> Faculty of Medicine Ain Shams Unversity 2015

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LIST OF ABBREVIATIONS

6WMD	6 walk minute distance
ACE	Angiotensin converting-enzyme
ACOS	Asthma-COPD overlap syndrome
AECOPD	Acute exacerbation of chronic obstructive pulmonary disease
AHR	Airway hyper-responsiveness
AO	Airway obstruction
ATS	American Thoracic Society
B cells	B lymphocytes
BAL	Bronchoalveolar lavage
BHR	Bronchial hyper-responsiveness
BODE index	a.Body-mass index, airflow Obstruction, Dyspnea, and Exercise
CD3+	Cluster of differentiation 3
CD ₄ +	cluster of differentiation 4
CD8+	Cluster of differentiation 8
CO	Carbon monoxide
COPD	Chronic obstructive pulmonary disease
Cox-1	Cyclo-oxygenase-1
CT	Computed tomography

DLCO......Diffusion capacity of the lung to carbon monoxide

ELISAEnzyme-linked immune-sorbent assay

FEV1.....Forced expiratory volume in the first second.

FEV1/FVC...The percentage of the vital capacity which is expired in the first second of maximal expiration

FVC.....Forced vital capacity

GERD.....Gastro-esophageal reflux disease

GINA.....Global Initiative for Asthma

GM-CSFGranulocyte macrophage colony stimulating factor

GOLDGlobal Initiative for Chronic obstructive lung disease

 H_2O_2Hydrogen peroxide

HRQoLHealth-related quality of life

ICAM-1.....Intercellular adhesion molecule

ICSInhaled corticosteroids

IFN- γ Interferon- γ

IgA.....Immunoglobulin A

IgE.....Immuno-globulin E

IL-4Interleukin 4

LABALong-acting beta2-agonist

LAMALong-acting muscarinic antagonists

MCP-1.....Monocyte Chemoattractant Protein-1

MHCMajor histocompatibility complex

MIPMacrophage inflammatory protein

mMRC Modified Medical Research Council.

mRNAMessenger Ribonucleic acid

NAEPP EPR-3 National Asthma Education and Prevention Program Expert Panel Report 3

NIPPV......Non invasive positive pressure ventilation

NONitric oxide

NODNon-obstructive disease

NOS.....Nitric oxide synthase

NPV.....Negative pressure ventilation

 O_2 -Super oxide anions

OADs.....Obstructive airway diseases

OH.....Hydroxyl radicals

PaCO₂......Partial pressure of carbon dioxide in arterial blood

PAFPlatelet-activating factor

PaO₂.....Partial pressure of oxygen in arterial blood

PDGFPlatelet derived growth factor

PEF.....Peak expiratory flow.

PGD₂Prostaglandins D₂

PGE₂.....Prostaglandins E₂

ROIReactive oxygen intermediate

RSV.....Respiratory synctial virus

SaO₂Oxygen saturation in arterial blood

SRH.....Self-rated health

T cellsT lymphocytes

 $TGF-\beta$Transforming growth factor beta

TH0 cellsNaïve T-cells

TH1 cells T helper type 1

TH2 cellsT helper type 2

Th-2.....T-helper 2 cells

TMB.....Tetra-methyl-benzidine

TNF.....Tumor necrosis factor

TNF- αTumor necrosis factor- α

VCAM-1 Vascular cellular adhesion molecule

VCDVocal cord dysfunction

VIPVaso-active intestinal peptide

VLA-4very late antigen-4

WHO......World Health Organization

YLDSYears of living disabilities

NTRODUCTION

Asthma and chronic obstructive pulmonary disease (COPD) are a major public health problem because of their high and still rising prevalence, their associated morbidity, mortality and socioeconomic costs (Bahadori et al., 2009; GOLD, 2011).

Asthma is recognised as an allergic disease that develops in childhood, characterised physiologically by reversible airflow obstruction, and has an episodic course with a generally favourable prognosis, responding well to anti-inflammatory treatment. In contrast, COPD is typically caused by tobacco smoking, develops in mid to later life and is characterised by incompletely reversible airflow limitation that results in a progressive decline in lung function leading to premature death (Gibson and Simpson, 2009).

Although asthma and COPD are different diseases, differential diagnosis is sometimes difficult and may be impossible in some older patients (Guerra, 2004).

Furthermore, asthma and COPD may coexist: more than 40% of patients with COPD report a history of asthma, and asthma has been recognized to be a risk factor for developing COPD. Patients who have both COPD and asthma (overlap syndrome) have a more rapid disease progression, a worse

health-related quality of life, more frequent respiratory exacerbations, increased co-morbidities and health care utilization than those with either disease alone (Roberto de Marco et al., 2013).

Overlap syndrome is recognized by the coexistence of increased variability of airflow in a patient with incompletely reversible airway obstruction. Patients typically have inflammatory features that resemble COPD, with increased airway neutrophilia, as well as features of airway wall remodeling. Overlap syndrome can develop when there is accelerated decline in lung function, or incomplete lung growth, or both. The risk factors for these events are shared, such that increasing age, bronchial hyper-responsiveness, tobacco smoke exposure, asthma and lower respiratory infections/exacerbations are significant risk factors for both incomplete lung growth and accelerated loss of lung function. Studying these events may offer new insights into the mechanisms and treatment of obstructive airway diseases (Gibson and Simpson, 2008).

AIM OF THE WORK

To evaluate the prevalence of asthma- chronic obstructive pulmonary disease (overlap syndrome) in Embaba Chest Hospital.

Chapter (1):

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Definitions:

COPD is a preventable and treatable disease state characterized by airflow limitation which is progressive, not fully reversible, associated with airway inflammation, hyper responsiveness and systemic manifestations including skeletal muscle dysfunction, cachexia, cardiovascular and osteo-skeletal alteration. The airflow obstruction is generally due to chronic bronchitis or emphysema (ATS, 2007).

Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease with some significant extra pulmonary effects that may contribute to the severity in individual patients. Its pulmonary component is characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases (GOLD, 2010).

This definition does not use the terms chronic bronchitis and emphysema and excludes asthma (reversible airflow limitation).