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AAT	alpha one antitrypsin
ACE inh.	Angiotensin converting enzyme inhibition
AECOPD	acute exacerbation of chronic obstructive pulmonary disease
ANA	antinuclear antibody
ANOVA	analysis of variance
ANP	atrial natriuretic peptide
ARDS	adult respiratory distress syndrome
ATS	American thoracic society
AVP	arginine vasopressin
BAL	bronchoalveolar lavage
BMD	bone minerals density
BMI	body mass index
BMRC	British medical research council
BODE method	body mass index ,obstruction, dyspnea and exercise method
BTS	British thoracic society
C3	complement 3
CAP	community acquired pneumonia
CLK	collagen like region
CMV	cytomegalovirus
COPD	chronic obstructive pulmonary disease
CRP	C-reactive protein
CSF	cerebrospinal fluid
CXC	cystein-x-cystein
CXR	chest X-ray

DNA	deoxyribonucleic acid
DPI	dry powder inhaler
ECG	electrocardiogram
EGF	epidermal growth factor
ERS	European respiratory society
ESR	erythrocyte sedimentation rate
ET-1	endothelin-1
ETS	environmental tobacco smoke
FEV1	forced expiratory volume in one sec.
FFM	fat free mass
FVC	forced vital capacity
GM-CSF	granulocyte- macrophage coloney stimulating factor
GOLD	Global Initiative For Chronic Obstructive Pulmonary Disease
H2O2	hydrogen peroxide
HAP	hospital acquired pneumonia
HCC	hepatocellular cell carcinoma
HRCT	high resolution computed tomography
HS	highly significant
IC	inspiratory capacity
ICAM-1	intercellular adhesive molecule 1
IgG	immunoglobin G
IL-8	interleukin 8
iNOS	inducible nitric oxide synthase
LTB4	leukotriene B4
LVRS	lung volume reduction surgery

Mac-1	macrophage associated antigen-1
MAF	macrophage-activating factor
MCP-1	macrophage chemotactic protein-1
MDI	metered dose inhaler
mEPHX1	microsomal epoxide hydrolase X1
MIF	migration-inhibitory factor
MIP-1B	macrophage inflammatory protein -1B
MMP	matrix metaloprotenase
NE	neutrophil elastase
NICE	National Institute For Clinical Excellence
NIV	non-invasive mechanical ventilation
NKcells	natural killer cells
NO	nitric oxide
NO.	number
NS	non significant
PC groups	phosphocholine groups
PC	phosphocholine
PCO2	partial pressure of carbon dioxide
PDE4 inh.	Phospphhhhhodiesterase-4 inhibitory
PDGF	platelet derived growth factor
PEF	peak expiratory flow
PGE	prostaglandin E
PH	pulmonary hypertension
PMNL	polymorphy nuclear leukocytes
PO2	partial pressure of oxygen
RAAS	rennin-angiotensin-aldosteron system

SaO2	oxygen saturation
SD	standard deviation
SHBG	sex hormone binding globulin
SLE	systemic lupus erythromatosis
SNS	sympathetic nervous system
TGF-B	transforming growth factor-B
TGF-B1	transforming growth factor beta 1
TLC	total lung capacity
TLR	toll- link receptor
TNF	tissue necrotizing factor
UTI	urinary tract infection
VA/Q scan	ventilation / perfusion scan
VAP	ventilatory acquired pneumonia
VHS	very highly significant
VIP	vasoactive intestinal peptide

Introduction

The chronic obstructive pulmonary disease is the major cause of the morbidity worldwide, It is predicted to become the fourth leading cause of death and disability worldwide by year 2020 **(Gold,2006).**

The chronic obstructive lung disease is preventable and treatable disease with some significant extrapulmonary effects that may contribute to the severity in individual patients and its pulmonary components is characterized by airflow limitation that is not fully reversible and The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases **(Soriano,2005).**

It is classified according to the symptoms , spirometric findings and presence of complications and the acute exacerbation of COPD are characterized by a changes in the patient`s baseline as dyspnea ,cough or/and increased sputum amount **(Soriano,2005).**

Also the exacerbation frequency increases with the severity and is associated with poorer health outcomes **(Donaldson,2006).**

The C-reactive protein is a member of the Pentraxin family of proteins and discovered by Tillett and Francis in 1930, it is an acute phase protein predominantly produced and secreted by hepatocytes , other cells including lymphocytes ,kupefer`s cells ,monocytes and macrophages can also produced C-reactive

protein **(Castell, 2000).**Normally, there is no C-reactive protein detectable in blood and positive test means inflammation in body due to different causes ,where the induction of C-reactive protein synthesis is triggered by a number of cytokines chiefly IL-6 ,which is released from a variety of cell types and mainly from macrophages and monocytes at inflammatory sites **(Gabay,1999).**

Also, the C-reactive protein secreted from local respiratory tract and damage of lung function in COPD patients is associated with the increase of C-reactive protein level **(Zhong,2005).**

The C-reactive protein measurements provide prognostic information beyond that achieved by traditional markers of prognosis in patient with mild to moderate COPD and enable more accurate detection of patient at high risk of mortality. Although, The C-reactive protein level are increased in COPD but it is not certain ,whether they are associated with adverse clinical outcomes**(Anthonisen,2006).**

Dev and Colleagues noted that positive C-reactive protein play an important role in an acute exacerbations of COPD and bacterial culture is not the most dependable factor of an acute exacerbations of COPD **(Dev,1998).**

AIM OF THE WORK

To Study the changes of serum C-reactive protein level in patients with an acute exacerbations of COPD and stable COPD patients, and the relationship of that to pulmonary function levels and arterial blood gases .

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Chronic Obstructive Pulmonary Disease (COPD) is a major cause of chronic morbidity and mortality throughout the world. Many people suffer from this disease for years and die prematurely from it or its complications. COPD is the fourth leading cause of death in the world**(WHO,2000).**

Chronic obstructive pulmonary disease can be regarded by describing & each of its words as follows:

Obstructive: partly blocked

Pulmonary: in the lungs

Disease: sickness

(GOLD, 2002)

Definitions:-

The Global Initiative for Chronic Obstructive Pulmonary Disease **(GOLD, 2008)** defined COPD as a preventable and treatable disease with some significant extrapulmonary 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.

The definition of COPD has been always a concern to chest physicians. It was changed several times from the first trials in 1819, when Laennec first described the pulmonary catarrh as an inflammation of the mucous membrane of the bronchi and emphysema as an increase in the size of the airspaces in the lungs **(Fletcher et al., 1976)** to the latest GOLD (global initiative for obstructive lung disease) definition During the past half of this century, definitions of Chronic Obstructive Pulmonary Disease have been refined repeatedly as the result of recent clinical observations, new diagnostic tools and sophisticated epidemiologic studies **(Fishman et al., 1998).**

In 1819 Laennec described "pulmonary catarrh" as an inflammation of the mucous membrane of the bronchi causing abundant secretion of mucus. Laennec also said that emphysema was "an increase in the size of the airspaces in the lungs" and this anatomical description is similar to the modern definition **(Fletcher et al., 1976).**

The Ciba Symposium (1959) defined chronic bronchitis as chronic or recurrent cough together with expectoration, which occurred "on most days for at least three months in the year during at least two years".

The American Thoracic Society (ATS) in 1995 defined COPD as:

a disease state characterized by the presence of airflow obstruction due to chronic bronchitis or emphysema, the airflow obstruction is generally progressive and may be accompanied by airway reactivity and may be partially reversible.

The European Respiratory Society (ERS) in 1995 defined COPD as: a disease characterized by reduced maximum expiratory flow and slow forced emptying of the lungs, which is slowly progressive and mostly irreversible to present medical treatment.

The National Institute for Clinical Excellence (NICE) in 2004 defined COPD as: a disease characterized by airflow obstruction which is usually progressive, not fully reversible, and does not change markedly over several months. The disease is predominantly caused by smoking.

Other attempts at defining COPD with reference to spirometric or functional criteria were undergone e.g. The BTS functional definition: " Chronic obstructive pulmonary disease (COPD) is characterized by airflow obstruction defined as a reduced FEV1(forced expiratory volume in 1 second) and a reduced FEV1/FVC ratio (where FVC is forced vital capacity), such that FEV1 is less than 80% predicted and FEV1/FVC is less than 0.7.

The airflow obstruction is usually progressive, not fully reversible and does not change markedly over several months. The disease is predominantly caused by smoking**(BTS, 2004).**

It was always believed that COPD is comprised of two conditions namely: Chronic bronchitis and Emphysema.

Chronic bronchitis is defined clinically as the presence of a chronic productive cough for 3 months in each of 2 successive years, provided that other causes of chronic cough have been ruled out **(ATS, 1995).**