الملخص العربي

ان مرض السدة الرئوية المزمنة هو مرض مزمن ينتج عن انسداد الشعب الهوائيه نتيجه التدخين المستمر ,والتدخين هو عامل مشترك بين مرضي السده الرئويه المزمنه ومرضي الشرايين التاجيه وهو ايضا اساسي في تشخيص مرض السده الرئويه لذاللك ان عمليات تغيير الشرايين التاجيه من العمليات الاساسيه في مرضي قصور الشرايين التاجيه .

ان عمليات تغيير الشرايين التاجيه لها العديد من التحضيرات التي تسبق العمليه وهي من العمليات التي تحتاج الي متابعه بعد العمليه نظرا لخطورتها علي المرضي.

يوجد ارتباط وثيق بين مرضي السدة الرئويه المزمنه وبين مرضي قصور الشرايين التاجية, توجد اربعة اسباب لهذه العلاقه منها تشابه الجينات الوارثيه بين الاثنين,والالتهابات الشديده التي يسببها مرض السدة الرئويه المزمنه,تمدد الحويصلات الهوائيه في الرئتين وارتفاع ضغط الشريان الرئوي.

هذه الرساله تمت لمعرفه تاثير عمليات جراحه تغيير الشرايين التاجيه علي مرضي السدة الرئوية المزمنة ودراسه المضاعفات الناتجه عن العمليه.

تبينت من احصائيات هذه الرساله انه يوجد قصور كبير في وظائف التنفس لهولاء المرضي منها انخفاض كبير في مخزون الهواء من الرئه وانخفاض في كميه الهواء داخل الحويصلات الهوائيه الصغيرة ووجود ارتفاع ملحوظ في وظائف الكبد ووجود بعض التغييرات في غازات الدم بعد العمليه.

تاثير هذه العمليه علي مرضي السده الرئويه المزمنه كان نتيجه الالتصاقات في عظمه القص, وقصور في عمل عضله الحجاب الحاجز, وتغيير كبير في تبادل الغازات في الشعب الهوائيه , ووجود المريض علي ماكينه القلب الصناعي يعمل علي تراكم البصاق بشكل كبير والتصاق الشعيبات الهوائيه الصغيره وعدم وصول الدم بشكل منتظم الي العضلات.

تم تسجيل العديد من المضاعفات لمرضي السده الرئويه المزمنه منها الانسكاب البلوري الذي يشمل

40% من الحالات,الالتهابات الرئويه في 8% من الحالات,تضخم متزايد في غضله القلب, وتزايد حده مرض السده الرئويه المزمنه وهذه المضاعفات قد تجعل المريض بحاجه للمكوث في الرعايه المركزه لفترات اطول وقد تصل الي التهاب رئوي نتيجه جهاز التنفس الصناعي والذي قد يتسبب في وفاه المريض بنسبه قد تصل الي 75 % من هذه الحالات وقد يحدث ارتشاح رئوي في بعض الحالات نتيجه ضعف في عضله القلب والتي تتطلب ووجود المريض علي جهاز التنفس الصناعي لفترات اطول.

يوصي بعمل اعاده تاهيل لهؤلاء المرضي عن طريق الحركه المبكرة للمرضي والذي يساعد عي تحسن وعي المريض وحالته الذهنية وعدم تعرضه لجلطات القدم ,وتنشيط كهربائي للعضلات وكذالك استخدام بعض الاجهزه التي تساعد علي زياده قدره الرئه علي استيعاب الهواء وتحسن وظائف التنفس.

تبين من الرساله انه يوجد قصور في وظائف التنفس لمرضي السدة الرئوية المزمنة وضعف في القدره الاستعابية للرئتين ووجود مضاعفات عديده لهذا النوع من العمليات ,وان عمليات تغيير الشرايين التاجيه لها تاثير طويل علي مرضي السدة الرئوية المزمنة لذللك ينصح بعمل وظائف تنفس لهولاء المرضي قبل وبعد العمليه وعمل تاهيل طبيعي لهولاء المرضي.

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Last, but not least, I want to express my cordial feelings towards my dear professors in our department, to whom I owe a lot of things more than I can count.

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ASCVD	Atherosclerotic cardiovascular vascular disease.
ACB	Aorta coronary bypass.
ARDS	Acute respiratory distress syndrome.
BLVR	Broncosopic lung volume reduction.
BMI	Body mass index.
CABG	Coronary artery bypasses grafting.
CPB	Cardiopulmonary bypass.
COPD	Chronic obstructive pulmonary disease.
CAD	Coronary artery disease.
DD	Differential diagnosis.
DVT	Deep venous thrombosis.
ECG	Electrocardiogram.
FVC	Forced vital capacity.
FEV1	Forced expiratory volume in the 1st second
ICU	Intensive care unit.
IS	Incentive spirometry.
IMA	Internal mammary artery.
KFT	Kidney function test.
LAD	Left anterior descending.
LFT	Liver function test.
LIMA	LeftInternal mammary artery.
MV	Mechanical ventilation.
NIPPV	Noninvasive positive pressure ventilation.
NEMS	Neuro muscular electrical stimulation
PO2	Arterial oxygen tension.
PCO2	Arterial carbon dioxide tension.
PTCA	Percutaneous trans luminal coronary angiography.
PFT	Pulmonary function test.
PTI	The percentage thickness increase.
RCA	Right coronary artery.
SFL	Smoking free legislation.
SaO2	Arterial oxygen saturation.

Chronic Obstructive Pulmonary Disease

Definition:

COPD is a mostly preventable and treatable disorder characterized by expiratory airflow limitation that is not fully reversible.

The air flow limitation is often progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases especially cigarette smoking. [1, 2]

Epidemiology:

COPD is a major cause of morbidity and mortality worldwide. [3] Although the prevalence of COPD is difficult to determine is estimated to affect about 24 million American. [4]

COPD and other chronic lower respiratory tract represent the third leading cause of death in the United States. Chronic obstructive pulmonary disease is projected to rank as the fifth worldwide burden of disease by 2020. [5]

Epidemiology in Egypt:

In Egypt there is about 3.77 million COPD patient according to the last survey done in Egypt in 2010[6].

Etiology of COPD:

COPD occurs in 10-20 % of smokers indicating that there is probable genetic susceptibility.

Exposures to:

1- Tobacco smoke: Cigarette smoking is by far the most important risk factor for COPD either active or passive exposure to smoke as well as fetal affection in pregnant females**.** [7]

2- Occupational dusts and chemicals: When the exposures are sufficiently intense or prolonged, such as those experienced by miners in many countries. [8]

3- Outdoor and indoor air pollution: Although the issue is not settled, available evidence suggests that air pollution might be a factor in the development of COPD. [9]

Genes:

Alpha 1-antitrypsin deficiency is found in 1-2 of COPD patients clinical characteristics of affected patients may include a minimal smoking history, early onset COPD younger than 45 years old also a family history of lung disease or lower lobe predominant. [10]

Pathophysiology of COPD

Processes in the lungs and airways are important in the pathogenesis of COPD include:

- Mucous gland hyperplasia particularly in the large airways, with mucous gland hypersecretion and therefore a chronic productive cough is produced.[11]

- Mucosal damage from smoke, squamous metaplasia and replacement of the normal ciliated columnar epithelium by squamous epithelium as shown in figure (1)

- Chronic inflammation and fibrosis of small air way characterized by CD8 lymphocyte, Macrophage and Neutrophils infiltration with release of pro-inflammatory cytokines.[12]

- Emphysema due to alveolar wall destruction causing irreversible enlargement of air spaces distal to the terminal bronchioles with subsequent loss elastic recoil and hyperinflated lungs.[13]

- The thickened pulmonary arteriolar wall and remolding occurs with hypoxia leads to increased pulmonary vascular resistance, pulmonary hypertension and impaired gas exchange. (Barbera et al., 2003).

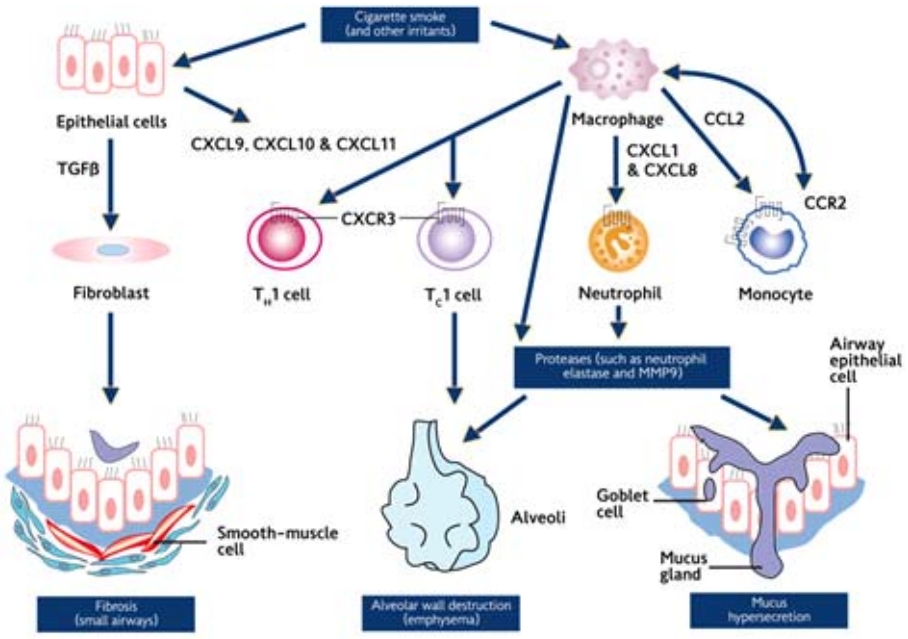

Figure (1): shows Pathophysiology of COPD

Clinical aspect of COPD:

- **COPD** is a heterogeneous disease: two main phenotypes of the disease are recognized **chronic bronchitis and emphysema.**
- **Chronic bronchitis** is characterized by cough and sputum production for 3 months in each of two consecutive years.[15]
- **Emphysema** is defined as destruction of the airways distal to the terminal bronchioles and gradual destruction of the alveolar septa and the pulmonary capillary bed.[14]
- The symptoms may precede the development of air flow limitation by many years. Inflammation and secretion provide the obstructive component of the disease, in contrast to emphysema chronic bronchitis is associated with relatively undamaged pulmonary capillary. The body responds by decreasing ventilation and increasing cardiac output (ventilation -perfusion mismatch) leading to hypoxia, polycythemia and increased carbon dioxide retention and eventually these patients develops signs of right sided heart failure.[16]

COPD Severity according to NICE Guidelines:

- **Mild**: FEV1 50-80% predicted may or may not be symptomatic.

- **Moderate:** FEV1 30-49 % Predicted, Increased FRC, Reduced DLCo likely to be symptomatic (cough, sputum, dysponea).

- **Severe:** FEV1 <30% predicted with marked hyperinflation, TLCO usually low with signs of corpulmonale.

Classification based on post bronchodilator FEV1:GOLD2014

- **Mild** FEV1>80 % predicted.
- **Moderate** 50%< FEV1 < 80%.
- **Severe** 30%<FEV1< 50%.
- **Very severe** FEV1 < 30% .

Clinical features:

History:

Patient exposed to risk factor such as smoking and occupational or environmental exposure. [38]

Symptoms:

- **Cough:** chronic cough, usually the first symptom of COPD to develop,

intermittent at the beginning then becoming progressive throughout the day.[18]

- **Sputum:** initially occurs in the morning but later will be present all day long. It is usually tenacious and mucoid and in small quantities. A change in sputum color (purulent) or volume suggests an infective exacerbation.[19]
- **Hemoptysis:** it is not uncommon during exacerbation; however the presence of blood should always alert the physician to the possibility of alternative diagnosis.[20]
- **Dyspnea:** is usually progressive and over time it becomes persistent. It is the reason most patients seek medical attention and is a major cause of disability and anxiety associated with the disease.[21]
- **Wheezing and chest tightness**: are relatively non-specific, in stage II, stage III and stage IV particularly during exercise.
- Weight loss may occur with end stage COPD.

Signs:

Depend on the severity of the underlying disease:

- Raised respiratory rate.
- Barrel shaped chest.
- Resonant bare area of the heart.
- Use of accessory muscle of respiration.

- Quite breath sound.
- Signs of cor-pulmonale and co2 retention (ankle oedema, raised juglar venous pressure, plethoric conjunctiva and polycythemia).[22-23]

Extra pulmonary aspects of COPD

Cardiovasular diseases

- Ischaemic heart disease.
- Hypertension.
- Pulmonary hypertension.
- Congesive heart failure.

Metabolic disorder

- Osteoporosis.
- Skeletal muscle weakness.
- Cachexia: weight loss and muscle wasting.
- Diabetes mellitus.
- Metabolic syndrome.

Other co morbid diseases:

- Lung cancer.
- Chronic kidney disease.