STUDY OF PREVALENCE OF DEPRESSION AND ANXIETY IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND ITS RELATION WITH SMOKING

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ABSTRACT

The psychiatric manifestations of medically ill patients have been accredited for decades.. However COPD patients are really a challenge to the healthcare system, constituting a considerable proportion of persons with long-term diseases.. In recent years, attention has been focused on the results that COPD patients undergo distress like anxiety and depression. Earlier examinations have exposed with COPD, almost 50% of patients are ill from a clinical diagnosis of anxiety and/or depression(Bacon, Lavoie, & Laurin, 2012).Moreover, in patients who have been already diagnosed with COPD, the most important precautionary measure in hindering the decline of pulmonary function is stoppage of smoking (Scale, n.d.).Even though cigarette smoking is the prime risk factor for chronic obstructive pulmonary disease have not been fully studied. Since cigarette smoking is found to be associated with both depression /anxiety disorders and COPD, it is also possible that the link in between is because of the mediation or confounding by cigarette smoking.Recently a study has revealed that over 65% of cigarettes are being used by persons with mental disorder.(Jenkins et al., 2012) There is also some evidence in the literature for a dose–response relationship between anxiety/depressive disorders and cigarette smoking.(Jenkins et al., 2012)) Since the major cause of this disease is exposure to cigarette smoke, chronic obstructive pulmonary disease is largely preventable.In view of the fact that the chronic obstructive pulmonary disease (COPD) is an almost irreversible clinical condition, the aim of treatment in COPD patients is not to cure but to reduce the symptoms, increase their functioning capacity, and improve the quality of life of the patients(Article, 2002). Indeed depression is going to be a major load to the health care expenditure around the planet. ("Overseeing Anxiety and Depression in Patients) In brief, COPD is a chronic multisystem disorder with comorbities and cigarette smoking is a foremost danger factor for its occurrence.

Interplay of several factors such as gender, educational, socioeconomic background, and marital status also do contribute to psychiatric co-morbidity like depression and anxiety in COPD. Unfortunately, neglected in COPD patients attending the outpatient department in our Indian set upThe following study is conducted to evaluate COPD patients attending general hospital outpatient department for the presence of depression and anxiety and several contributing risk factors

REVIEW OF LITERATURE
Prevalence of Psychiatric Co-morbidity in COPD:

Thas been established from various studies that depression has a prevalence of 10% when compared with other psychiatric disorders. Depression has a point prevalence of 2.3% to 4.9% in the general population. And the surprising fact is up to 70% to 80% of this depression is treated by non psychiatrists or they are untreated (Goldstein, 2008)

Going through the studies that have assessed feelings of anxiety in these patients describe prevalence in 2–96% levels. Generalized anxiety disorder accounts 10% – 33% while the panic state is between 8% and 67%. (Goldstein, 2008)

First before going into a detailed discussion about the psychiatric manifestations of COPD, it is important to know certain facts about the disease and how smoking is related to the disease per se.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE:

These processes mark and progress to the hallmarks of the clinical picture of COPD:Airflow limitation and,Dyspnea or shortness of breath,Cigarette-associated deleterious agents impair lung defense mechanisms leading to a progressive airflow limitation. (Lira- mandujano et al., n.d.)

Spirometry:

It measures the *FEV1- forced expiratory volume in one second (the maximum volume of air that can be exhaled out in the first second of a large breath). This instrument also measures FVC -the forced vital capacity (the maximum volume of air that can be exhaled out in a completely large breath (OMS).* (Lira-mandujano et al., n.d.)

The diagnosis of COPD is made when the FEV1/FVC ratio is less than 70%.

PATHOPHYSIOLOGY of COPD:

Cigarette-associated deleterious agents will injure the epithelium lining the airways and will trigger the key process that causes specific airway inflammation plus structural changes associated with smokers and continue to progress in spite of smoking cessation. In another key phenomenon is increased oxidative stress. (Update, 2009) Several studies have documented chronic obstructive pulmonary disease is because of the changes upsetting small airways and lung parenchyma.The reduction in FEV1 is mostly related to thickening of the walls of tiny conducting airways.

Oxidative stress and protease–antiprotease imbalance in COPD:

Oxidative metabolism is over-activated in chronic obstructive pulmonary disease. The most important outer source of oxidants is cigarette smoke. The inflammation of bronchi concerning phagocytes, such as macrophages and neutrophils, leads to an internal production of oxidants. Antioxidants like the haemoxygenase (HO)-1 pathway and the glutathione system may neutralize oxidative stress.

Proteases are formed by a variety of cells within the airways. Their activity is being synchronized with the manufacture and release of antiproteases, such as alpha -1 antitrypsin, tissue inhibitor of metalloproteinases (TIMPs) and secretory leukoprotease inhibitor. Cigarette smoke nullifies the activity of antiproteases .The phagocytes are a chief supply of proteases; the macrophages from COPD patients are found to be less capable to release TIMPs in reaction to stimulation. Proteases cause elastin along with collagen destruction and then to destruction of the tissue that characterize emphysema.

Role of Inflammatory cells in COPD:

Neutrophils are granulocytes that are related with COPD and their number is enlarged in sputum, broncho alveolar lavage and smooth muscle of sufferers. The elevation in neutrophil can lead to an amplified discharge of oxidants and proteinases that perpetuate imbalance which causes lung destruction.

Smoking causes the pooling and activation of macrophage cells

Cigarette smoke exposure also activates dendritic cells. This may add to neutrophilic inflammation and decreased protective reaction to infection by viruses that lead to periodic exacerbations in these patients. B-lymphocytes are essential agents in the adaptive immune system. T- lymphocytes are found in the airways; together with predominantly CD8+ cells associated with the reduction in pulmonary function.(Update, 2009)

Structural changes in COPD:

The permanent airflow limitation which is the key feature of COPD might be associated with structural changes. The bronchial epithelium is modified in the proximal and smaller airways.

The hallmarks of smoking induced COPD are:

- Squamous cell metaplasia and Goblet cell hyperplasia.

Enlargement of bronchial glands,Increase in number of bronchial glands,increase in smooth muscle mass All these evidences of remodeling have been direct detrimental effect of the smoking (Update, 2009)

Clinical Consequences of COPD:

The most comprehensive measure of COPD impact is apparently health-related QOL.. Dyspnoea is linked to both respiratory (defective gas exchange, hyperinflation etc.) and non- respiratory (including **depression**, etc.) features of COPD. (Update, 2009)

Further it may be speculated that the chronic occurrence of aversive dyspnoea over the course of COPD cause changes in brain areas with significance for emotions processing (e.g. limbic system structures), thus sensitizing these areas towards the processing of anxiety and depression, preferentially.

COMORBIDITIES IN COPD:

.Few of the important chronic illnesses and their potential consequences are listed below.

Extra-pulmonary disease -Possible consequences in COPD patients:

1. Cardiovascular disease,Cancer (including lung cancer)Increased mortality Increased dyspnoea,osteoporosis,Depression,Diabetes,obstructive sleep apnea

SMOKING IN COPD:

The literature reveals that 80 to 90% of chronic obstructive pulmonary disease is an effect of smoking. The treatment possibilities are narrow in COPD,besides,smokingcessation.

CONSULTATION -LIAISON PSYCHIATRY:

The Indian scenario shows reference from various specialties including medicine with psychiatric disorders like anxiety and depression is present in about 40 to 50% of the patients. Though the Indian published data is limited, most tertiary hospitals in India carry out liaison work with various departments like neurology, organ transplant, intensive care units and cosmetic surgery so as to give comprehensive health services to patients (Parkar & Sawant, 2010.

DEPRESSION AND ANXIETY IN COPD:

In a developing country like India, often the psychiatric co morbidity with chronic physical illness goes undetected. (Sharma et al., 2013) Often patients report that breathlessness as the highly disabling feature. Anytime during the exacerbations of the disease, dyspnoea varies with the disease severity. Patients frequently express an uncontrollable and highly distressing anxiety that was associated with severe breathlessness. A state of either frustration or irritability is considered as a precipitator of anxiety that in turn leads to feeling of breathlessness.

Anxiety in COPD patients is leading to increased problems in areas of marital life and family interactions. Commonly seen in male sufferers are a decreased level of efficacy, which may even be just a subjective state, to cope with the symptoms of the disease and it is also noted that poor social support is associated with anxiety. (Goldstein, 2008) Females are observed to report greater level of anxiety and more reduced ability to do daily activities. (Laurin, Lavoie, Bacon, Dupuis, & Lacoste, n.d.)According to the DSM-IV, depressive mood is often accompanied with tears, easy irritability, anxiety, ruminations, fearfulness and worrying in bodily wellbeing. Hence depression problems in COPD may be difficult to diagnose if they are attributed to the underlying lung disorder.Apathy and reduced self worth are often present in patients with various stages of COPD. The prominent features of depression in these patients might also interfere with the choice of antidepressants therapy. (Goldstein, 2008)Most studies demonstrated low HQoL in COPD patients with depression and anxiety. Moreover, depression showed strong correlations with HQoL when compared to spirometric measurement of lung function. **Causes and Risk Factors for Depression and Anxiety in COPD:**

● lower family income,more stressful life events,higher education levels.Furthermore, it might be assumed that there might be a common linking path between COPD, anxiety, and depression. Several authors have identified that cigarette smoking might be a common pathway as it   is associated with both psychiatric morbidity, as well as in the development of COPD.

Testosterone activates the serotonergic system in various parts of the brain including frontal cortex, cingulated cortex and hippocampus region. It also inhibits monoamine oxidase-A (MAO-A),. These observations have a significant implications for the hypothesized causal link between reduced levels of testosterone and development of depression.(Halabi et al., 2011)

Potential Mechanisms

Simply it is a bidirectional interaction between depression and factors associated with COPD some factors were found to be connected to a greater chance of contracting depression like female gender, lower economic classes, active smoking and long term O_2 therapy(Fraternity, Hospital, & Kaneko, 2012

Biological Pathways

Anxiety and depression are coupled with unnecessary excitation of the HPA axis and amplified inflammatory response in the bodily systems which may perhaps be accountable for an augmented risk of illness exacerbations. In diseases of the cardiac system, it has been adequately acknowledged that continuous activation of the sympathetic part of the autonomic nervous system (SNS) and activity in excess of normal in the hypothalamo-pituitary–adrenal (HPA) axis (through inflammatory mechanisms) can throw in to the evolution of many of the heart disease risk factors noted in depressed sufferers . (Bacon et al., 2012)

Psychiatric disorders, especially depression, can provoke immune deficiencies and swell the chance of post infarction deaths which makes us suppose that these disorders (depression) can have an eventual influence on COPD.

The spotlight has largely be on four factors:(Mikkelsen et al., 2004)Hypoxia,Smoking,Exacerbations andUntreated depression,

All of these issues might in turn direct to the occurrence of secondary depressive state and neurocognitive deficits in patients with COPD. In addition to the contribution of developing psychomotor dullness and memory disturbances, hypoxia is also known to cause depressive phenomenon.This, resulting in neuropsychological deficits predominantly impaired attention and memory functioning.Smoking in these patients has two different effects. It is known to be both anxiolytic and anxiety provoking, and in a big population section, Thus smoking is noticed to a have a significant causative role in many ways.(Mikkelsen et al., 2004)he ensuing impairment in QOL of the sufferers is allied with bigger impediment of daily activities and a disabling mood state. (Mikkelsen et al., 2004)

Cognitive and Behavioral Mechanisms

Patients with emotional problems often suffer from little self- esteem or self-efficacy, which may contribute to worsened disease, linked coping abilities and impaired self-care, implying the refusal to get involved in pulmonary rehabilitation strategies, lessened physical doings, failure or inability to leave smoking, deprived eating behavior, and poor devotion to treatment plans. To understand better, it has been revealed that having a low body mass index (18.5 kg/m2) bears an imperative risk factor for inpatient–managed exacerbations even in stable disease state. (Bacon et al., 2012)

Literature have showed the need to have phenotypical division of COPD by multidimensional assessment, to determine looks of disease and exacerbation "behavior" among individuals as well as groups of patients.(Bacon et al., 2012)

Recent ECLIPSE cohort study too emphasized a similar verdict to search for those combinations of disease attributes which are interconnected with anxiety and depression, and which ones bestow increased risk for exacerbations. This would help clinicians to better identify those patients with better prognosis among all patients with COPD. (Bacon et al., 2012).

Psychological and physiological explanatory models

1. **The hyperventilation model** considers that dyspnea and panic crop up from the same clinical presentation which is called the *"hyperventilation syndrome"*.
2. **The carbon dioxide hypersensitivity model** is from the observations that in 2/[3rd] of patients distressing from panic, lactate is the cause for panic anxiety, appear to produce a *''suffocation false alarm''*.
3. **The cognitive behavioral model** is formulated on the assumptions that fear and misapprehension of bodily experiences emerging as a

result of breathlessness and hyperventilation are catalyzing a panic reaction.

Coping Strategies in COPD:

Researchers have proposed a *'theory of stress and coping'* based on cognitive understanding, which has had an enormous impact on the literature about chronic illness. **Coping** defines the relentlessly changing behavioral and cognitive pains to master lessen or bear the inner and/or outer demands which are produced by a stressful situation.

Self-efficacy theory Simply, it is the degree of the faith that one has on his capability of carrying out an exact behavior.

Smoking –depression relationship:

Identifying COPD patients on the verge of developing depression would facilitate the control of an important co morbidity conferring an additional risk for poor outcomes.The somatic symptoms of depression include anorexia, loss of weight, hypersomnia/insomnia, lassitude and psychomotor retardation, all of which may equally be a consequence to the medical illness. Nonetheless, the consensus is that it is better to apply the diagnostic criteria in psychiatry without any adjustment, but being alert of the risk of over diagnosis, so that all symptoms are measured including whatever their cause may be. It is important that the evaluation of patients focuses on the cognitive domain of depression. Depression is known to cause an individual to begin substance abuse, especially smoking as a self- medicating strategy. Then this smoking is known to reduce the physical activity of the persons.(Atlantis, Fahey, Cochrane, & Smith, 2013) There is a two way relationship between depression and sleep disturbances and this has to be considered in evaluating an elderly individual with COPD presenting with insomnia.(Ito et al., 2012)

A liable clarification is that smokers with a record of depression or current depression experience nicotine withdrawal features with added intensity in the post termination period, depression-susceptible smokers are much less likely to stay abstemious from smoking (Scale, n.d.)

The Impact of Depression

The presence of co morbid depression in COPD patients disapprovingly influences the respiratory function through a multifaceted interplay of exogenous factors such as age of the individual, disease severity and level of dyspnoea with internal factors like perceived health status, exercise capacity, and personal weariness.

. Many small studies have reported that psychiatric morbidity might contribute to a higher level of influence on patients' quality of life than the core symptoms of the disease by itself.. (Out-, 2006)

In addition, the presence of co-morbid depression has been connected with a reduced perceived quality of life (QOL) in COPD patients, which also interrupts marital relations.(Norwood, n.d.)

The burden of this disease has risen in the recent past among many of the eastern countries because of the escalating tobacco use among public.

Smoking Cessation and COPD:

Smoking cessation is the most effective intercession in stopping the evolution of COPD, as well as increasing survival and controlling morbidity. This is why smoking cessation should be the apex priority in the management of COPD. Smokers who quit before they turn 35 yrs can anticipate a life expectancy comparable to non smokers.

studies imply that smokers are not constantly recognized or treated. (Cornuz & Willi, 2008). It is made a verdict that the concrete number of attempts to stop smoking by an individual is a seer marker of his/ her *"desire to quit".*

Recently investigations estimating the concentration of nicotine and cotinine, an important metabolite have been invented. Cotinine plasma concentration, with a longer half life than nicotine is taken as a more reliable measure of dependency. A concentration of 15 ng / ml is taken as a non smoking level and persons with chronic smoking might show a value in the range of 200 ng / ml to 1000 ng / ml.. In fact, even lighter

smoking (5 cigarettes/day) have been coupled with eminent health risks, especially with view to lung cancer.(Jiménez-ruiz, 2008)

Methods intended to slow but sure reduction devoid of drugs against quitting ''cold turkey'' have shown to lead to unrelenting craving and protracted withdrawal symptoms. Smokers compensate by taking more and/or deeper puffs per cigarette when they try to reduce their smoking. Lifestyle modifications and aerobic exercise activities were known to attenuate the psychiatric morbidity in chronic lung disease patients. (Coventry et al., 2013)

Guidelines promote that physicians pursue the **''5 As''** in beginning assessment and involvement with tobacco users.1}Asking if he or she have tobacco: document tobacco use status for every patient.2}Advising to stop smoking.3}Assessing the promptness to quit tobacco use.4}Assisting the patient in their quit effort.5}Arranging for follow-up contacts and relapse prevention motives.(Cornuz & Willi, 2008)

Above all, since even the most valuable intervention is overwhelmed by relapse, the physician should notify the patient that the characteristic smokers require several serious and valiant cessation efforts before ultimately achieving enduring success. setbacks shouldn't be looked as failures and should be looked upon as learning experiences.

With each relapse, the patient is pushed forward in a position to learn more on the subject of his/her individual strengths and vulnerabilities. In this background this study is intended to examine the co-morbid anxiety and depression in COPD patients, in a view to expose the importance of this proven problem as well as investigate its relation with smoking.

AIM

The aim of this study is to study the prevalence of depression and anxiety in patients with chronic obstructive pulmonary disease.

OBJECTIVES

- To measure the prevalence of depression and anxiety in patients with chronic obstructive pulmonary disease in a hospital based population
- To investigate the impact of depression and anxiety on the illness and quality of life of patients
- To study the influence of depression and anxiety on the smoking behavior

METHODOLOGY

SAMPLE – 60 male patients with COPD are selected into the study after meeting the study criteria.

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60 normal and healthy subjects from general population

STUDY DESIGN

Cross sectional study with case-control design

MATERIALS USED

A. Semi structured proforma to elicit socio demographic and other related data like smoking status, cumulative pack years, duration of illness, family history of mental illness, disease pattern, treatment history and clinical history,MMSE,MINI plus,Hamilton depression rating scale,Hamilton Anxiety rating scale,WHOQOL-BREF questionnaire

Motivation to Stop Smoking scale (MTSS)

CASES

60 stable male patients with COPD attending the outpatient department, are selected into the study after meeting the study criteria.

INCLUSION CRITERIA

- Patients with COPD diagnosed by chest physicians
- Age – 30 to 60 yrs old
- Gender – males
- Clinically stable with no change in medication in the past one month and no hospital admission in past 6 weeks
- Willing to provide informed consent

EXCLUSION CRITERIA

- Patients with psychiatric illness or treatment before the study
- Patients with substance dependence other excluding nicotine
- Patients with other significant pulmonary disease
- Patients with chronic non pulmonary disease
- Patients on long term treatment with steroids
- Unwilling and uncooperative patients

CONTROLS

Roughly age matched 60 normal and healthy individuals are selected from the general population

INCLUSION CRITERIA

- Normal healthy subjects from general population
- Age 30 to 60 yrs
- Male gender
- Willing to provide informed consent for the interview

SCALES

HAMILTON DEPRESSON (HAM – D) SCALE

The current version of HAM-D lists 21 items of which one the first 17 are scored.

HAMILTON ANXIETY (HAM-A) SCALE

This is another scale which is used to evaluate anxiety symptoms in the patient. It has got 14 items and the rater rates the individual on a 5 point scale for each of the 14 items. Seven denotes psychic anxiety and the left over seven items deal with somatic anxiety.The total score ranges from 0 to 56.

-BREF Questionnaire

It is a generic health related questionnaire developed by the WHO QOL group - which helps to quantify the health related quality of life. It consists of 24 facets and it gives outline of scores on 4 dimensions of QOL.

After calculating the total of each domain the raw scores are converted in to transformed scores using the tables. The First transformation converts raw domain scores to a range of 4 to 20 and the second transformation converts it to a 0 to 100 scale.

MTSS – MOTIVATION TO STOP SMOKING SCALE

It is imperative to note that this evaluation explicitly includes intention, desire and belief into a single item with the anticipation that this will give the most cost-efficient likely measure**.** It is a quantitative device for population surveys along with studies about the effect of modalities designed at mounting motivation to end smoking. (Kotz, Brown, & West, 2013)

MINI – PLUS

It is *the Mini International Neuropsychiatric Interview*. It was structured to diagnose the axis I psychiatric disorders according to the DSM IV and ICD 10. It is a structured interview that is brief and used by clinicians after giving appropriate instructions and training the patients about it

MINI MENTAL STATE EXAMINATION (MMSE)

It is a 30 point questionnaire by Folstein. It is used to assess

➢ Cognition,Orientation to time and place,Memory,Language use including comprehension/word repetition / reading / writing / drawing, attention, and arithmetic ability.

Any score more than 27 out of 30 is considered normal. In the study, the MMSE is used to screen the patients and assess if there is any cognitive impairment in them to confirm that they are able to understand the questions asked and participate in the study. Any patients with scores below cut off and clinically evident cognitive deficits are excluded from the study.

TYPE OF THE STUDY

Case – control, cross sectional study

PERIOD OF THE STUDY

Sep 2018 to Dec 2018

PLACE

Out Patient Department of Thoracic medicine,govt.theni medical college,Theni.

METHODOLOGY

The study was conducted in the OP Department of Thoracic Medicine from Sep 2018 to Nov 2018. 60 stable patients with physician diagnosed chronic obstructive pulmonary disease and staged according to GOLD staging were taken into the study. Random sampling method was used to reduce any impact of sampling bias. Similarly 60 age matched participants from the general population were taken as control group.

In view of the effect of hypoxia secondary to the physical illness and additionally may be due to smoking, there can be chances of cerebro- vascular diseases and so cognitive functions were screened in the participants. It was ensured that the controls were healthy and did not suffer from either chronic physical or mental illness.Also recent medicine change and acute exaberation are excluded.

Female patients with COPD were not taken into the research since we wanted to know the relationship between smoking and outcome variables. The patients attending for regular follow up were screened and interviewed with the semi- structured proforma which is explained below. Before enrolling the patients in the study, detailed verbal and written information was given to the patient regarding the aim and the protocol of the study and a signed informed consent was obtained.

Socio demographic information collected included,

1. Age,Addres,sEducation,Occupation,Socioeconomic status,Marital status

Family type,The study included patients in the age range of 30 to 60 years of age since COPD was reported to be common in that age group. Controls were taken in a matching pattern though exact matching was not done.,The educational status was recorded in five groups,The occupation status of the individuals were divided in to five groups as,,The income was recorded in four groups, according to Kuppusamy classification; it is recently revised to seven groups such as

1. less than 1520, 2. 1521 to 4555, 3. 4556 to 7593, 4. 7594 to 11361,

5. 11362 to 15187 ,

6. 15188 to 30374 and

7. More than 30374.

● The marital status of the individuals was collected as married, unmarried and Divorced / widowed
● The family structure of the individuals was recorded as nuclear type, joint family and living alone.

The history and details pertaining to the physical illness collected to correlate clinical parameters with depression and anxiety were as follows:

1. Smoking status – the current smoking status of the individuals as current smoker, smoker in the past (ex-smoker) and as never a smoker
2. Cumulative smoking in pack years –

No. packs of cigarettes smoked per day X no. of years the person has smoked. It is the unit for measuring the amount a person has smoked over a long period of time.

3. Body mass index as weight (Kg) / Height (m^2) and it was recorded as underweight, normal, overweight and obese.
4. Number of illness exacerbations in the last one year and that was classified into
a. Less than or equal to 2 and
b. More than two.
5. Duration of diagnosed illness as in years
6. Whether hospitalized for the most recent exacerbation : Yes / No
7. History of any other chronic medical illness is screened to exclude them since it might have an individual correlation with the outcome variable.
8. History of any other substance use / abuse is taken to exclude them
9. Family history of psychiatric illness as Yes / No
10. Current Treatment history records as antibiotics, β agonists, steroids and as a combination of drugs.

       Thorough general and systemic clinical examination was done for each person. Then the patients were subjected to pulmonary function tests. Pulmonary function tests using Spirometry were done on every individual with the assistance of health care workers trained in that procedure who were available in the college. $FEV_{1 \%}$ and FEV_{1}/ FVC ratio were calculated from the spirometry assessment. Patients were then classified into various stages following GOLD guidelines (Global Initiative for Chronic Obstructive Lung Disease).

GOLD SPIROMETRIC CRITERIA:

FEV1/FVC < 0.7 in every stage
- MILD –FEV1 ≥ 80% predicted
- MODERATE – 50% ≤ $FEV_{1 \%}$ < 80% predicted
- SEVERE - 30% ≤ FEV1 < 50% predicted
VERY SEVERE - FEV1 < 30% predicted or FEV1 < 50% predicted plus chronic respiratory failure

       Then brief psychiatric history and short Mental State Examination were done. Rating scales mentioned in the previous section were applied to the individuals.

Statistical Analysis

       The statistical analysis was carried out using SPSS-20 software. The collected data were summarized using proportion, number, percentage, mean, and standard deviation, whichever was found to be appropriate for the particular data. The independent student T test was applied to compare continuous variable and chi square test was applied to compare categorical variables. One way ANOVA test was used for more multiple comparisons. In all statistical analysis, 2-sided tests were used for the appropriate data and results were considered statistically significant, if P value is less than 0.05.

       More than 90 percent (91.7%) of the population are married. 70 percent of the total sample had completed at least higher secondary level. Most population belonged to middle socio- economic strata (4556 – 7593). 73.3 percent of the control persons lived as nuclear family compared to 60% in the cases. Nearly 2/3 (70%) of the cases were doing either semi-skilled or skilled employment. Chi square test was applied to compare the proportions between cases and controls.

       P value was significant only for the comparison of socio-economic status

COMPARISON BETWEEN HAM A AND HAM D TABLE: 1

HAM D	GROUPS				Total	
	Case		Control		N	%
	N	%	N	%		
Normal	34	56.7	55	91.7	89	74.2
Mild	20	33.3	5	8.3	25	20.8
Moderate	3	5.0	0	.0	3	2.5
Severe	3	5.0	0	.0	3	2.5
Total	60	100.0	60	100.0	120	100.0
Chi-Square Test			P-Value			
Fisher's Exact Test			**<0.001**			

Table: 1 give the comparison between proportions of cases and control group in their HAM D scores. 43.3% of those with the illness have positive HAM D scores (mild – 33.3%, moderate – 5% and severe – 5%) while only 8.3% of the controls have positive HAM D scores and all belonged to mild scores. P value was statistically significant. (<0.001)

TABLE: 2

COMPARISON OF HAM A BETWEEN CASES AND CONTROLS

HAM A	GROUPS				Total	
	Case		Control		N	%
	N	%	N	%		
Normal	46	76.7	57	95.0	103	85.8
Mild	13	**21.7**	3	**5.0**	16	13.3
Moderate	1	1.7	0	.0	1	.8
Total	60	100.0	60	100.0	120	100.0
	Chi-Square Test		P-Value			
	Fisher's Exact Test		**0.007**			

Table: 2 give the comparison between proportions of cases and control group in their HAM D scores. 43.3% of those with the illness have positive HAM D scores (mild – 21.7% and moderate – 1.7%) while only 5% of the controls have positive scores and all were in the mild category.

The mean age in cases is 49.92 and for men in the control group is 48.48. There was a significant difference (P< 0.001) in the data about cumulative pack years of smoking between the cases and controls mean being 24.23 and 10.57 respectively.

ANOVA table: 3
One way ANOVA to compare the mean values between HAM D levels

Variable	Sum of Squares		df	Mean Square	F-Value	P-Value
Pack years	Between Groups	4556.184	3	1518.728	7.000	**<0.001**
	Within Groups	12150.549	56	216.974		
	Total	16706.733	59			
Duration	Between Groups	320.758	3	106.919	10.632	**<0.001**
	Within Groups	563.175	56	10.057		
	Total	883.933	59			
AGE	Between Groups	72.991	3	24.330	.757	0.523
	Within Groups	1799.592	56	32.136		
	Total	1872.583	59			

Table 3 give the analysis of the relationship between quantitative variables of age, pack years and duration of illness and HAM D scores. ANOVA analysis depicts a significant relationship between duration of illness and pack years of smoking and HAM D scores in the comparison between cases and controls (P< 0.001)(df – 3).

ANOVA- Table 4
One way ANOVA to compare the mean values between MTSS

Variable	Sum of Squares		df	Mean Square	F-Value	P-Value
AGE	Between Groups	169.783	6	28.297	.881	.515
	Within Groups	1702.800	53	32.128		
	Total	1872.583	59			
Pack years	Between Groups	5268.717	6	878.119	4.069	**.002**
	Within Groups	11438.017	53	215.812		
	Total	16706.733	59			
	Between Groups	102.655	6	17.109	1.161	.341

Duration	Within Groups	781.279	53	14.741		
	Total	883.933	59			

Table: 4 give the analysis of the relationship between quantitative variables of age, pack years and duration of illness and MTSS scores.

ANOVA analysis depicts a significant relationship between pack years of smoking and MTSS scores in the comparison between groups (P< .002)

The correlation between the quality of life (QOL) and pack years   and duration of illness were compared and shown in **Table 6**.it shows that the there is strong correlation (P value <0.001) of duration of illness and QOL in all the domains [D1, D2, D3, D4] and good correlation (P value < .025) between pack years and domains 1 and 4 of QOL.

One way ANOVA to compare mean values between marital statuses, education, socio-economic status, family type, occupation, smoking status,MI, exacerbation of illness and QUALITY OF LIFE did not achieve any statistical significance.

ANOVA – Table: 5

One way ANOVA to compare mean values between GOLD levels

		Sum of Squares	df	Mean Square	F-Value	P-Value
D1	Between Groups	2975.152	2	1487.576	7.098	**.002**
	Within Groups	11945.181	57	209.565		
	Total	14920.333	59			
D2	Between Groups	2847.921	2	1423.961	5.368	**.007**
	Within Groups	15119.729	57	265.258		
	Total	17967.650	59			
D3	Between Groups	3478.255	2	1739.127	6.346	**.003**
	Within Groups	15620.595	57	274.046		
	Total	19098.850	59			
D4	Between Groups	3105.876	2	1552.938	7.524	**.001**
	Within Groups	11765.057	57	206.405		
	Total	14870.933	59			

One way ANOVA to compare mean values of QOL domains   between disease severities found a significant association between all four domains of QOL and staging (GOLD) and severity of the disease as indicated by FEV1% .

ANOVA Table: 6
One way ANOVA to compare mean values between Treatments

		Sum of Squares	df	Mean Square	F-Value	P-Value
D1	Between Groups	4556.444	3	1518.815	8.207	**.000**
	Within Groups	10363.889	56	185.069		
	Total	14920.333	59			
D2	Between Groups	3227.616	3	1075.872	4.087	**.011**
	Within Groups	14740.034	56	263.215		
	Total	17967.650	59			
D3	Between Groups	3397.046	3	1132.349	4.038	**.011**
	Within Groups	15701.804	56	280.389		
	Total	19098.850	59			
D4	Between Groups	4777.085	3	1592.362	8.834	**.000**
	Within Groups	10093.848	56	180.247		
	Total	14870.933	59			

Table: 6 ANOVA to compare mean values of QOL domains with treatment found a significant association ($P < .05$; df – 3) between all four domains of QOL and treatment options of the disease as from table 6.

Table: 7

Treatment	HAM D								Total	
	Normal		Mild		Moderate		Severe			
	N	%	N	%	N	%	N	%	N	%
Antibiotics	4	11.8	0	.0	0	.0	0	.0	4	6.7
Beta agonists	25	73.5	13	65.0	0	.0	0	.0	38	63.3
Steroids	4	11.8	6	30.0	0	.0	1	33.3	11	18.3
Combination	1	2.9	1	5.0	3	100.0	2	66.7	7	11.7
Total	34	100	20	100	3	100	3	100	60	100

	Chi-Square Test	P-Value
	Fisher's Exact Test	**0.001**

From **table: 7** it is known that 63.3% are on beta agonists drugs and 11.7% are receiving a combination of drugs. 65% of those who are mildly depressed are on beta agonists and three patients are severely depressed with two [n=66.7%] receiving combination of drugs. There was a significant association between treatment and HAM D within cases. ($P<.05$)

Statistical tests to compare the proportions of other socio-demographic variables like marital status, socio-economic status, occupation among those with positive HAM A scores in the total cases did not achieve

significant P value (i.e., P < 0.05)

DISCUSSION

The aim of the present study was to assess the levels of depression and anxiety and to explore the influence of the disease on the quality of life of the patients having COPD. The present study used HAM-D, HAM-A and WHOBREF questionnaire to evaluate depression, anxiety and the quality of life of the subjects respectively while an Indian study done in Aligarh they employed Beck's Depression Inventory and Hamilton Anxiety scale. (Mohammad et al., 2012

From the various studies it was observed that the prevalence of depression in this group of COPD patients ranges from 7% to 71% and that of anxiety is 9% to 42%(Maurer et al., 2008)

☒ In this study we found that the proportions of diseased who are having depression are 43.3% in comparison with 8.3% in controls. Anxiety was found in 23.4% of patients with only 5% of controls showing anxiety scores. Majority of them were having milder levels

of depression and all those who were anxious were in the milder level. These findings are in the same line with that of previous

studies showing a higher prevalence of depression and anxiety compared to controls.

☒ In a systematic review of previous studies, the prevalence of depression in moderate-to-severe COPD patients ranged 7–42%,

which is also supported by subsequent studies. Hospitalizations further elevated these proportions. (Goldstein, 2008)

☒ Also we achieved a significant relationship between quality of life patients and the severity of the disease as given by duration of illness

and GOLD staging, with severe stages showing a corresponding inverse relationship with QOL which was similar to a previous study from China (Lou et al., 2012).

☒ In our study we found a good interrelation between the disease factors of cumulative pack years, the length of the disease process and the level of depression. This indicates that both smoking and the disease process had effects on the depressive symptoms independently. The relationship between depression and smoking have been explained in previous works ("Effect of Treating Depression on Quality-of-Life and Exercise Tolerance in Severe COPD," 2005).

It was also found that the effect of pack years and duration of the illness were significantly associated with HAM D scores only in the milder levels than in the severe depression levels. This is an important finding because it is the milder levels of depression that ought to be frequently get unnoticed and overlooked.

☒ The cumulative effect of smoking was additionally related the levels of motivation to stop smoking as explored in many earlier researches studying the motivation to stop smoking in COPD patients, leading to quick disease development and also, more risk for admission–treated exacerbation(Bacon et al., 2012).

☒ This relationship was also achieved in our study revealing that there is an inverse relationship between levels of depression & anxiety and the level of motivation to stop smoking. We could observe from the scatter plots that as the severity of either depression or anxiety raised, the motivation level tends to dip down. So they are less likely to abstain from smoking, accelerating the progress of the disease once again.

☒ Those patients in the initial stages of the disease process were found to be depressed compared to later stages (nearly 90%). This might be

viewed as a result of an adaptive mechanism to the onset of chronic disabling disease.

☒ Additionally we found an association of the treatment types the patients were receiving with that of the anxiety scores. This might be viewed as an effect of the drug, especially sympathomimetics causing anxiety symptoms. We also worked to find a relationship between the disease status and stage of the disease and the motivation level of the patients to stop smoking.

Though we did not get a statistical significance, we could interpret that the reduced levels of motivation on patients were seen in those patients who are in the initial stages of the disease (i.e., Stage 2 and Stage 3). This is an important concern because it reveals the importance of enhancing the motivation of these patients in the initial stages of the disease.

Compared to the general community, tobacco use in the form of smoking is observed to be more (approaching 50%) in persons with psychiatric illness. Certainly this elucidates the subsequent finding of ineffective attempts to cease from smoking and tobacco related morbidity in these persons(Stockings et al., 2012).

The helpfulness of smoking cessation programs is interfered by the co-occurrence of psychiatric disorders(Year & Review, 2013). So it should be clear that treatment of co-occurring psychiatric morbidity should be a part of the management strategies.

he importance of assessing the motivation of the individual and simultaneous treatment of depression and/or anxiety which will have its own impact on the motivation.

LIMITATIONS

The study was done in a hospital based population and being a hospital based study, finding of this study may not be generalized and it may not give the actual proportion of depression among patients with COPD of the whole country. It was a cross sectional study.

Likewise, diagnosis and the staging of COPD was made by Spirometry test plus the results were mixed from various examiner's involvement and directions given to the patients.

CONCLUSION & FUTURE PERSPECTIVES

The co-morbidity of depression and anxiety in COPD has been known for a long time. The higher prevalence of depression and anxiety in COPD and its impact on QOL have been acknowledged again through this study.The approximate measure of depression in COPD in this subcontinent is somewhere between 2.4% to 22%. remains either undiagnosed or under diagnosed.. This emphasizes the role of the psychiatrist to work in alliance with the physician to efficiently treat this chronic disease. Above all, national management guidelines for the depression among COPD patients should be developed and implemented(Bhowmik, Adhikari,Choudhury, & Ahmed, n.d.). A broad approach leading to valuable execution of a management plan is desired that yet again marks the value of liaison psychiatry.(Andenaes, 2005)

BIBLIOGRAPHY

- Article, O. (2002). Risk of depression in patients with chronic obstructive pulmonary disease and its determinants, (1), 412–417.

- Article, O., & De, S. (2010). Prevalence of Depression in Stable Chronic Obstructive, *102*(8).

- Atlantis, E., Fahey, P., Cochrane, B., & Smith, S. (2013). Bidirectional Associations Between Clinically Relevant Depression or Anxiety and COPD: A Systematic Review and Meta-analysis. *Chest*, *144*(3), 766–77. doi:10.1378/chest.12-1911

- Bacon, S. L., Lavoie, K. L., & Laurin, C. (2012). Pulmonary Perspective Impact of Anxiety and Depression on Chronic Obstructive Pulmonary Disease Exacerbation Risk, (April 2011). doi:10.1164/rccm.201105-0939PP

- Cornuz, J., & Willi, C. (2008). interventions in clinical practice, 187–191. doi:10.1183/09059180.00011003

- Coultas, D. B., & Edwards, D. W. (2007). Predictors of Depressive Symptoms in Patients with COPD and Health Impact, (March), 23–28. doi:10.1080/15412550601169190

- Fraternity, T., Hospital, M., & Kaneko, T. (2012). Depression in Japanese Patients with Chronic Obstructive Pulmonary Disease . Disease . A Cross- Cross - S ectional Study, (C). doi:10.4187/respcare.02065

- Goldstein, R. (2008). Anxiety and depression in end-stage COPD, *31*(3), 667–677. doi:10.1183/09031936.00125707

- Halabi, S., Collins, E. G., Thorevska, N., Tobin, M. J., & Laghi, F. (2011).
- Relationship Between Depressive Symptoms and Hypogonadism in Men with COPD, (708), 346–353. doi:10.3109/15412555.2011.594465

- Laurin, C., Lavoie, K. L., Bacon, S. L., Dupuis, G., & Lacoste, G. (n.d.).

- Sex Differences in the Prevalence of Psychiatric Disorders and Psychological Distress in Patients With COPD *. doi:10.1378/chest.07-0134

- Leventhal, A. M., Ph, D., Zvolensky, M. J., & Schmidt, N. B. (2011).

- Smoking-Related Correlates of Depressive Symptom Dimensions in Treatment-Seeking Smokers, *13*(8), 668–676. doi:10.1037/a0012570 Lipowski, Z. J. (1986). Consultation-liaison psychiatry: the first half century. *General Hospital Psychiatry*, *8*(5), 305–315.

- Lira-mandujano, J., Míguez-varela, M. C., & Cruz-morales, S. E. (n.d.).
- Psychological Approaches to Increase Tobacco Abstinence in Patients with Chronic Obstructive Pulmonary Disease< : A Review.
- Mikkelsen, R. I. E. L., Middelboe, T., & Pisinger, C. (2004). Anxiety and depression in patients with chronic obstructive pulmonary disease (COPD). A review, (1). doi:10.1080/08039480310000824
- Parkar, S. R., & Sawant, N. S. (2010). Liaison psychiatry and Indian research. *Indian journal of psychiatry*, *52*(Suppl 1), S386–8. doi:10.4103/0019-5545.69274

- Update, E. R. (2009). Recent advances in COPD: pathophysiology, respiratory physiology and clinical aspects, including comorbidities, *18*(114), 198–212. doi:10.1183/09059180.00005509

- /Year, C., & Review, I. N. (2013). Smoking cessation and COPD, *22*(127), 37–43. doi:10.1183/09059180.00007212