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RESEARCH ARTICLE

A PROSPECTIVE STUDY OF THYROID DYSFUNCTION AND CLINICAL PROFILE IN COPD PATIENTS IN A TERTIARY CARE CENTRE

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ABSTRACT

Background: In COPD patients the systemic inflammatory cytokines were increased and these inhibits the synthesis or secretion of TSH and peripheral conversion of T4 to T3, this leads to endocrine dysfunction in the form of hypothyroidism. Hypothyroidism causes alveolar hypoventilation, upper airway obstruction, Diaphragmatic dysfunction and respiratory failure. Altered endocrine function worsen the clinical manifestations of COPD and increases exacerbation frequency. **Aims/Objectives**: To assess the prevalence of thyroid dysfunction and clinical features of thyroidin COPD patients and to measure the relationship with COPD severity. **Methods:** 100 patients were collected from NMCH Hospital Kota. undergo Pulmonary function test, for 3 times at every 15 minutes interval and best of 3 readings were taken. The Forced Vital Capacity (FVC), (FEV1), FEV1/ FVC ratio were recorded. Blood sample was sent to check thyroid profile. **Result:** Out of 100 cases male patients (n=78), female (n=22). Most of patients in the age group of 51-70 (n=80). Hypothyroidism found in (n=53). overt hypothyroidism in 38, subclinical in 15. The mean TSH value is 10.35±1.72 and P-value is 0.0021. The correlation coefficient of FEV1 and TSH is -0.8305. that is good negative correlation. Means hypothyroidism was found in more severe COPD patient. **Conclusions:** Hypothyroidism is present in COPD, especially in severe form of COPD. Hypothyroidism not only increases COPD exacerbation but also affects the quality of life. Hence COPD patients should be regularly monitored for abnormal thyroid function and managed accordingly to improve their quality of life.

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INTRODUCTION

Chronic Obstructive Pulmonary Airway Disease is a Preventable & Treatable Disease which is characterised by irreversible airflow limitation of progressive in nature [(1)] occurring due to chronic inflammatory response affecting the lung parenchyma & airways to noxious stimulants & various toxic pollutants in the atmosphere. [(2)] The two classic phenotypes of COPD are emphysema and chronic bronchitis.[(3)] Emphysema is defined as enlarged airspaces (alveoli) whose walls break down resulting in permanent damage to the lung tissue. Chronic bronchitis is defined as a productive cough that is present for at least three months each year for two years. [(4)]

Emphysema is the structural abnormalities that can limit airflow and can exist without airflow limitation in a significant number of people. Chronic bronchitis does not always result in airflow limitation.[(5)] The most common cause of COPD is tobacco smoking. [(6)]however, the Other causes like genetic, indoor and outdoor air pollution, exposure to occupational irritants such as dust from grains and cadmium and fumes. [(7-11)] Alpha 1 antitrypsin deficiency is the strongest genetic factor in the development of COPD especially Emphysema. [(12)] Emphysema predominantly involved lower lobes, which is usually pan lobular or pan acinar type. [(11)] In developing countriesindoor air pollution may contribute as a significant

risk factor. Common source are use of coaland biomass such as wood and dry dung as fuel for cooking and heating.[13] COPD prevalence is being recognized increasingly in all countries. [14]The Global Burden of Disease Study has projected that COPD will become the third leading cause of death worldwide by 2021. [5] The cardinal symptom of COPD is the chronic and progressive shortness of breath. [15] Shortness of breath is often most distressing symptom responsible for the associated anxiety and level of disability. Symptoms of wheezing and chest tightness associated with breathlessness can be variable over the course of a day or between days, and are not always present. [5] Chronic cough, which may or may not be productive of mucus as phlegm. [16] An acute exacerbation is a sudden worsening of signs and symptoms that lasts for several days. [17] The key symptom is increased breathlessness and other more pronounced symptoms are of excessive mucus, increased cough and wheeze. A commonly found sign is air trapping.[18] The usual cause of an exacerbation is a viral infection, most often the common cold. [19] however, bacterial infection may also responsible. [20] The most common bacterial infection is caused by Haemophilus influenzae.[21]

COPD is multisystemic disorder, may alter the other organ function. Endocrine abnormality is one of them. The mechanisms by which COPD alters endocrine function are hypoxaemia, hypercapnia, systemic inflammation and glucocorticoid administration. [22] In COPD patients the levels of systemic inflammatory cytokines such as IL-6, IL-1 and TNF-αare increased. [23]These cytokines can inhibit the synthesis or secretion of TSH, T3 and thyroid hormone-binding proteinsand can decrease the mRNA for the hepatic enzyme iodothyronine deiodinase type-1[24] an enzyme that converts T4 to T3.[25] Altered thyroid function can worsen the clinical manifestations of COPD through several mechanisms include abnormalities in control of breathing, decreases in respiratory and limb-muscle mass and function, worsening of respiratory mechanics, impairment of cardiac function and disorders of fluid balance. [26]Hypothyroidism may also cause alveolar hypoventilation, decreased lung volumes, upper airway obstruction, depression in respiratory stimulus, and respiratory failure. [27] Diaphragmatic dysfunction and myopathies can be seen in patients with hypothyroidism. Inspiratory and expiratory muscle strength is linearly related to the degree of hypothyroidism. The myopathic manifestations is due to the impaired expression of myosin heavy chains IIb or to impaired neuromuscular transmission and impaired muscle energy metabolism, resulting from a defect in glycogen breakdown or mitochondrial function. [28]this leads to decreased respiratory muscle function and exercise capacity. [29] Weakness correlates with the severity of hypothyroidism and is reversed by replacement therapy.

Aim and objective

- To Assess the Prevalence of Thyroid Dysfunction and Clinical Features of thyroid in COPD patients.
- To Measures the relationship between COPD severity with the thyroid abnormalities.

METHODS

Source of data-This study was done on December 2019 to July 2021inthe Department of Respiratory medicine GOVT. Medical college, Kota in the 100 adult COPD patients who came in Outdoor/indoor.

Study design-Prospective study

Patients who were include- who were diagnosed as COPD as per GOLD – GLOBAL INITIATIVE FOR CHRONIC OBSTRUCTIVE PULMONARY DISEASE.

Patients who were excluded- who were refuse to give consent to participate in the study, the Known patients of Hypothyroid & Hyperthyroid, Patients Underwent Thyroid Surgery, Patientson Medications that Interfere with TFT - Amiodarone, Iodine Preparations, Immunosuppressive Drugs, Patients who have Bronchiectasis and Cystic Fibrosis.

Data collection-Based on gold staging, COPD patients were selected from outdoor and indoor. Their written consent was taken.The history was elicited. Age, sex, height, weight was recorded. Thorough clinical examination was carried out. Patients were made to undergo pulmonary function tests by using Medspiror, for 3 times at every 15 minutes interval and best of 3 readings was taken. The Forced Vital Capacity (FVC), FEV1, FEV1/ FVC ratio were recorded. (FEV1 is Forced Expiratory volume at the end of 1 second). Blood sample was drawn and send for testing of thyroid profile. TSH, Serum T3, Serum T4, free T3, free T4 was calculated.

INTERPRETATION OF RESULTS

TSH - (0.3-5.5 µIu/ml)
Free T4 - (0.89 - 1.72ng/dl)
Free T3 - (2.4-4.2pg/ml)

Statistical Analysis: The statistical software SPSS V.16.0 was used for all analysis of the data & MS Word & Excel have been used to generate graphs, tables, etc. Statistical analysis was done by using percentages, mean values, standard deviation, chi-square test, anova t-test and proportion test, regression coefficient. A P-value <0.05 was considered as statistically significant, P-value >0.05 was considered as not significant.

RESULTS

In our study 100 patients were included. Out of 100 Male patients- 78, Female patients - 22. In our study most of patients in the age group of 51-70. (N=80/100). In out of 100 patients (n= 56) are smoker. Most of males among all smokers. Non-smokers are 44.And most of non-smokers are females. Mean Values of FEV1 in Mild 81.2±1.78, Moderate 62.5±7.35, Severe 44.88 ±4.73 and Very-Severe 28.2 ±0.83. Out of 100 cases, Hypothyroid was found in 53 patients. Overt hypothyroidism in 38 patients and Subclinical in 15. Rest of them are normal (n=47). Mean Value ofTSH in Overt Hypothyroidism and Subclinical is 10.35±1.72. (P value is 0.0021), Free T4 is 0.56±0.18. (P value is 0.0001), Free T3 is 1.68±0.49. (P value is 0.0023). Correlation Coefficient between FEV1 & TSH: -0.8305 (Good Negative Correlation), FEV1 & T4 0.6041(Good Correlation), FEV1 & T3 0.5755 (Moderate Correlation). Means as % predicted FEV1 decreases, TSH value increases. Hypothyroidism is associated with severity. Means found in more severe COPD cases.

Table No.1. Mean Values of FEV1 In Mild, Moderate, Severe and Very-Severe COPD

COPD	Total patients	Sex wise		Mean	SD	P value
		Male	Female			
Mild	5	4	1	81.2	1.78	0.0001
Moderate	65	50	15	62.55	7.35	0.0001
Severe	25	21	4	44.88	4.73	0.0021
V. Severe	5	3	2	28.2	0.83	0.001

Table 2. Mean Value of TSH, T3, T4 In A Study Profile

Parameter	Mean	SD	P value
SERUM TSH	10.35	1.72	0.0021
SERUM T3	1.68	0.49	0.0023
SERUM T4	0.56	0.18	0.0001

Table 3. Correlation Coefficient Between COPD Parameter (FEV1) & Thyroid Biochemical Parameters (FT4, FT3)

FEV1 VS TSH	-0.8305	Good Neg correlation
FEV1 VS T4	0.6041	Good Correlation
FEV1 VS T3	0.5755	Moderate Correlation

Correlation Coefficient between FEV1 & TSH: -0.8305 (Good Negative Correlation).
Means as % predicted FEV1 decreases, TSH value increases.
Hypothyroidism is found in more severe COPD cases.

Table 4. Mean and Standard deviation of thyroid symptoms in COPD

Symptoms	Finding in no of patients	Mean	SD	P value
Hoarse Voice	31	11.01	1.19	0.0001
Constipation	36	10.82	1.56	0.0021
Poor Appetite	25	10.95	1.42	0.004
Weight gain	15	10.72	1.51	0.0024
Heat Intolerance	16	10.92	1.05	0.0001

We also evaluate the thyroid symptoms. The can be hoarse voice, weight gain, constipation, poor appetite, heat intolerance etc. There mean and standard deviation of Hoarse voice 11.01±1.19, Constipation 10.82±1.56, Poor Appetite 10.95± 1.42, Weight gain 10.72±1.51, Heat Intolerance 10.92±1.05. The P value is <0.05 that is significant.

DISCUSSION

COPD affects the whole body and causing various systemic manifestation. however, Ourmain concern our thyroid abnormalities, that was found in the form of hypothyroidism in our study population. In our study, we included 100 patients. Majority of them are male (n=78), while females (n = 22) made up the minority. The male-to-female ratio is 3.5 to 1.The majority of the participants was in the age group of 51 and 70, accounting for 80% (n=80). Out of them 60% (n=48) patients were between the ages of 61 and 70. Singh lalit et al[30]- A total of 201 cases of COPD were evaluated. Among 201 patients, 60 were females and 141 were males. There was male predominance with the male to female ratio of 2.3:1. The mean age of COPD patients was 62.6 years. Majority of patients were in the age group of 61-70 years. Smoking is the most common risk factor of COPD. we take into consideration. In out of 100 patientsmokers are 56% (n= 56). Most of them are males. Non-smokers are 44 out of 100. Most of non-smokers are females. According to GOLD staging the patient were categories into Mild, Moderate, Severe and Very Severe group. In our study out of 100, Mild cases are 5% (n=5), moderate 65% (n=65), Severe are 25% (n=25) and very severe patients are 5% (n=5). In our study mild cases are 5,in out of 5 thyroid abnormality was not found in any patient.

In 65 moderate cases thyroid disease in 40% (n=26). Overt hypothyroidism in 13, Subclinical hypothyroid in 13. In 25 Severe cases hypothyroid in 88% (n=22). Overt hypothyroid in 80% (n=20), and Subclinical hypothyroid in 8% (n=2).In Very Severe cases hypothyroid in 100% (n=5). Overt hypothyroid in all cases. Severity of COPD linearly related to thyroid abnormality. As severity of COPD increases, risk for hypothyroidism was increases. The Prevalence of hypothyroid in our study was found as 53%(n=53), in mild 0%, moderate in 26%, severe 22% and very severe 5%.Overt hypothyroidism in 38 patients and subclinical in15.These result show consistency with result of other studies. Singh P Lalit et al[30]- total of 201 cases of COPD, 130 (64.6%) were observed to be having thyroid disorders. Hypothyroidism was diagnosed in 119 (59.2%) cases and hyperthyroidism in 11 (5.4%) cases. P-value of association of COPD and thyroid disorders was 0.213. Chaudaryshyam et al [28] Out of 171 patients, thyroid dysfunction was present in 43 patients. All of them were hypothyroid. The prevalence of thyroid dysfunction was 25%. In Stage A it was 20.5%, Stage B 25.7%, Stage C 23.4%, and in Stage D 30.4%. Thyroid dysfunction was associated with more frequent exacerbation. P-Value was 0.04, that is significant in all stages of COPD. R.Vijayaragavan et al [31] Out of 100 patients, prevalence of thyroid dysfunction was67% (n=67). Out of 67, hypothyroidism observed in mild category 0, in moderate 34, and in severe in 33 cases. Mean value of TSH is of significantly elevated in moderate & severe COPD patients with its p-value being significant (<0.001). Mean Value of TSH in our study is10.35±1.72 and P-Value is 0.0021. Mean value of Free T3 is 1.68±0.49 and P-Value is 0.0023. Mean value of Free T4 0.56±0.18and P Value is 0.0001.

R. Vijayaragavan et al [31] Mean Value of TSH was 12.28±2.28, Free T3 2.38±0.707, Free T4 0.384±0.146. P-Value was <0.001. Clinical evaluation was also done in COPD patients. the clinical features that suggestive of thyroid abnormality are hoarse voice, constipation, poor appetite, weight gain, menstruation abnormalities in female, goiter, heat gain and cardiac manifestations like bradycardia, narrow volume pressure fatigue etc. we also evaluate theses symptoms in COPD patients. The Mean value was calculated. Mean value of Hoarse voice is 11.01, Constipation 10.82, Poor appetite 10.95, Weight gain 10.72, Heat intolerance 10.92. The P value is <0.05, that is significant.

CONCLUSION

Thyroid dysfunction is a most prevalent non-pulmonary symptom of COPD.It affects a large number of COPD patients, particularly those with the most severe form of the disease (GOLD STAGE 3 and 4). As COPD progresses, it causes a variety of thyroid function abnormalities, including hypothyroidism. Hypothyroidism has a significant impact on respiratory dynamics and mechanics, leading to frequent exacerbations and respiratory failure. The methods through which COPD affects the endocrine function are hypoxia, hypercapnia and inflammatory mediators. Hypothyroidism that not only increases COPD exacerbation but also affects quality of life. Hence COPD patients should be regularly monitored for abnormal thyroid function (TSH, FT3, FT4) and managed accordingly to improve their quality of life.

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REFERENCES

1. Effectiveness of clinical pharmacist intervention on health-related quality of life in chronic obstructive pulmonary disorder patients - a randomized controlled study; A. Suhaj, M. K. Manu, M. K. Unnikrishnan, K. Vijayanarayana, C. Mallikarjuna Rao. Journal of Clinical Pharmacy and Therapeutics, Issue: 2016,1, 78-83
2.Vestbo J, Hurd SS, Agusti AG, et al. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. Am J Respir Crit Care Med. 2013;187:
3. Differential Diagnosis of Adult Asthma; Stephen A.Tilles. Medical Clinics of North America, Issue: 2006,1, 61-76
4. Respiratory effects of borax dust. D H Garabrant, L Bernstein, J M Peters, T J Smith, W E Wrigh, Occupational and Environmental Medicine, Issue: 1985,12, 831-837.
5. Gold Report 2021Global Strategy for Prevention, Diagnosis and Management of COPD: 2021 Report (PDF). Global Initiative for Chronic Obstructive Lung Disease. 25 November 2020. Retrieved 28 June 2021.
6. Cough in Chronic Obstructive Pulmonary Disease, Kian Fan Chung, Peter M. A. Calverley, Cough: Causes, Mechanisms and Therapy, 125-135
7. COPD causes - occupations and substances". www.hse.gov.uk. Retrieved 3 July 2021.
8. Torres-Duque CA, García-Rodriguez MC, González-García M (August 2016). Is Chronic Obstructive Pulmonary Disease Caused by Wood Smoke a Different Phenotype or a Different Entity? Archivos de Bronconeumologia. 425–31.
9. Gold Report 2021, pp. 8–14, Chapter 1: Definition and overview.
10. Air pollution exposure in cities — European Environment Agency". Retrieved 28 June 2021.
11. Kurmi OP, Lam KB, Ayres JG. Indoor air pollution and the lung in low- and medium-income countries. EurRespr J. 2012; 40: 239.
12. Alpha 1-atitrypsin deficiency: from genetic routes to bedside; BaibaLāce, AlvilsKrams, DidzisPilāns, AstrīdaKrūmiņa, Proceedings of the Latvian Academy of Sciences. Section B. Natural, Exact, and Applied Sciences, Issue: 2009,3,81-86.
13. Exposure to indoor and outdoor air pollution from solid fuel combustion and respiratory outcomes in children in developed countries: a systematic review and meta-analysis: Valentina Guercio, Iulia C. Pojum, Giovanni S. Leonardi, Clive Shrubsole, Alison M. Gowers, Sani Dimitroulopoulou, Karen S. Exley, Science of The Total Environment, Issue: 2021, 142187.
14. Roberto R, Antonio A, Jean B et al. Pocket Guide to COPD diagnosis, management and prevention: Global Initiative for Chronic Obstructive Lung Disease (gold) update 2010.
15. Chronic obstructive pulmonary disease: A guide for the primary care physician; Gautam Raju Mehta, Rafat Mohammed, Sarah Sarfraz, Tajwaar Khan, Khansa Ahmed, Mauricio Villareal, Dennis Martinez, Joy Iskander, Rachid Mohammed, Disease-a-Month, Issue: 2016,6, 164-187.
16. What is Bronchiectasis? American Journal of Respiratory and Critical Care MedicineIssue: 2017,8, P15-P16.
17. Time course and pattern of COPD exacerbation onset; Shawn D Aaron, Gavin C Donaldson, George A Whitmore, John R Hurst, Tim Ramsay, Jadwiga A Wedzicha, Thorax, Issue: 2011,3, 238-243.
18. Gold Report 2021, pp. 104–109, Chapter 5: Management of exacerbations.
19. How Viral Infections Cause Exacerbation of Airway Diseases; Patrick Mallia, Sebastian L. Johnston, Periodical: Chest, Issue: 2006,4,1203-1210.
20. Role of Infection; Kamen Rangelov, Sanjay Sethi, Clinics in Chest Medicine, Issue: 2014, 87-100.
21. Haemophilus Influenza Type b; Susan Shoshana Weisberg, Disease-a-Month, Issue: 2007, 459-462.
22. Cardiovascular Comorbidities in COPD Patients; Roever L, Resende ES, Translational Medicine, Issue: 2015,03.
23.Chronic Obstructive Pulmonary Disease: A Chronic Systemic Inflammatory Disease; Stephan F. van Eeden, Don D. Si, Respiration, Issue: 2007,2, 224-238.
24. Induction of Type 1 Iodothyronine Deiodinase to Prevent the Nonthyroidal Illness Syndrome in Mice; August 2006; Jingyu Yu.
25. Wouters EF. Local and systemic inflammation in chronic obstructive pulmonary disease. Proc Am Thorac Soc 2005

26. Laghi F, Adiguzel N, Tobin MJ. Endocrinological derangements in COPD. Eur RespirJ. 2009; 34: 975–96.
27. Effect of thyroid function on COPD exacerbation frequency: a preliminary study; SevincSarincUlasli, SerifeSavasBozbas, Zeynep EraymanOzen, Berna Akinci Ozyurek, Gaye UlubayMultidisciplinary Respiratory Medicine, Issue: 2013.
28. Prevalence of thyroid dysfunction in chronic obstructive pulmonary disease patients in a tertiary care center in North India; Shyam Chand Chaudhary, Tauhid Ahmad, Kauser Usman,Kamal Kumar Sawlani, Kamlesh Kumar Gupta,Ajay Kumar Verma, and D Himanshu Reddy-J Family Med Prim Care. 2018 May-Jun.
29. COPD and Thyroid Dysfunctions; Claudio Terzano, S. Romani, G. Paone, V. Conti, F. Oriolo, Lung, Issue: 2013,1: 103-109
30. Lalit Singh, Abhishek Jain, Anurag Agrawal, Rajeev Tandon, Hemant Kumar. A study of prevalence of thyroid disorders in chronic obstructive pulmonary disease patients at a tertiary care center in U.P. International Journal of Contemporary Medical Research 2016;3(5):1239-1242.
31. R. Vijayaragavan, Dr.A. Prabhu Md, A Prospective Assessment Study of Thyroid Dysfunction in Moderate to Severe COPD. IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 18, no. 5, 2019, pp 33-36.