International Journal of Research in Medical Sciences
Kamdar DJ et al. Int J Res Med Sci. 2017 May;5(5):1802-1807
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20171539

Original Research Article

A study of the clinical profile of 50 patients of COPD with correlation between clinical, radiological and spirometric evaluation

Deepali J. Kamdar*, Dharmesh Kumar Patel

Department of Tuberculosis and Respiratory Medicine, GCS Medical College, Hospital and Research Centre, Ahmedabad, Gujarat, India

Received: 30 March 2017
Accepted: 03 April 2017

***Correspondence:**
Dr. Deepali J. Kamdar,
E-mail: drdjkamdar_27@yahoo.com

ABSTRACT

Background: Chronic obstructive pulmonary disease (COPD) is a common, preventable and treatable disease that is characterised by persistent respiratory symptoms and airflow limitation that is due to airway and/ or alveolar abnormalities usually caused by significant exposure to noxious particles or gases. Tobacco smoking, occupational exposure to organic and inorganic dusts, chemical agents and fumes and biomass cooking are the risk factors for COPD. Chronic dyspnoea, cough, sputum production, wheezing and chest tightness are the common symptoms of COPD. The present study was undertaken to evaluate the clinical, radiological and spirometric parameters in patients with COPD and to demonstrate a correlation between them.

Methods: This was a prospective study of 50 patients of COPD who presented to out- patient department. We included all patients above 40 years of age with a smoking index of 200 or more, or history of exposure to occupational dust, biomass fuel gas or exposure to other obnoxious gases; and who had history of dyspnoea and cough. Spirometry was performed to confirm the diagnosis and to grade the severity of airflow obstruction. History of dyspnoea, cough, sputum production, wheezing, chest tightness, fever, weight loss and the number of exacerbations in the previous year was noted. We then performed a detailed clinical examination. Blood was sent for haemogram and arterial blood gas analysis and all patients underwent an ECG, 2- D Echo and HRCT of the thorax. We then studied the correlation between the clinical, radiological and spirometric profiles in these patients.

Results: Out of the 50 patients, majority was between 50-59 years of age, with male to female ratio of 1.94:1.00. History of smoking was present in 74% patients, exposure to biomass fuel in 12% and exposure to occupational dust in 6% patients. Commonest symptom was dyspnoea (in 100% patients) followed by cough (88%), sputum production (68%), wheezing (58%), chest tightness and fever (30%) and weight loss (28%). HRCT was positive in 75% patients, while ECG changes were seen in 42% patients and pulmonary hypertension was present in 54% patients. A significant association was observed between grade 5 dyspnoea on mMRC, hypoxia, hypercarbia, pulmonary hypertension and Gold-5 airflow obstruction.

Conclusions: In the present study of 50 cases, COPD was seen predominantly in male patients, with a mean age of presentation between 50- 59 years. Tobacco smoking was the commonest etiological factor. Clinical symptoms most commonly documented were dyspnoea, cough with or without expectoration, wheezing, chest tightness, fever and weight loss. A significant association was observed between grade 5 dyspnoea on mMRC, hypoxia, hypercarbia, pulmonary hypertension and Gold-5 airflow obstruction.

Keywords: Airflow obstruction, Dyspnoea, Hypoxia, Hypercarbia, Pulmonary hypertension, Risk factors

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a common, preventable and treatable disease that is characterised by persistent respiratory symptoms and airflow limitation that is due to airway and/ or alveolar abnormalities usually caused by significant exposure to noxious particles or gases.[1] The chronic airflow limitation is caused by mixture of small airways disease (obstructive bronchiolitis) and parenchymal destruction (emphysema). These changes do not always occur together, but evolve at different rates over time. Chronic respiratory symptoms may precede the development of airflow limitation and may exist in individuals with normal spirometry.[2,3] Across the world, tobacco smoking is the commonest risk factor for COPD.[4] Both active and passive smoking account for respiratory symptoms. Occupational exposure to organic and inorganic dusts, chemical agents and fumes, are other risk factors for COPD.[5,6] There is growing evidence that indoor air pollution from biomass cooking and heating in poorly ventilated dwellings is an important risk factor for COPD.[7] The role of outdoor air pollution is unclear. Chronic and progressive dyspnoea is the cardinal symptom of COPD and a major cause of disability in these patients.[8] A simple measure of dyspnoea is the Modified British Medical Research Council (mMRC) Questionaire.[9] The mMRC questionnaire relates well to other measures of health status and predicts future mortality risk.[10] Cough is often the first symptom of COPD. Cough may be productive or unproductive.[11] Cough with sputum production is present in 30% of patients. These symptoms may vary from day to day. Wheezing and chest tightness are other accompanying symptoms. Fatigue, weight loss and anorexia are common problems in patients with severe and very severe COPD.[12] Ankle swelling may be an indicator of corpulmonale. Spirometry gives an objective measurement of airflow limitation. A post bronchodilator FEV1/ FVC < 0.07 is an indicator of airflow obstruction. Based on the post bronchodilator FEV1, airflow obstruction is classified as Mild (Gold 1), Moderate (Gold 2), Severe (Gold 3) and Very severe (Gold 4).[1] The present study was undertaken to evaluate the clinical, radiological and spirometric parameters in patients with COPD and to demonstrate a correlation between them.

METHODS

This was a prospective study of 50 patients of COPD who presented to the department of Respiratory Medicine over a period of one year with a clinical history of COPD. COPD was considered in any patient who presented with dyspnoea, chronic cough or sputum production, and/ or a history of exposure to risk factors as per GOLD guidelines1. We included all patients above 40 years of age with a smoking index of 200 or more, or history of exposure to occupational dust, biomass fuel gas or exposure to other obnoxious gases; and who had history of dyspnoea and cough. We obtained an informed consent of all the included patients. The presence of a post- bronchodilator FEV1/FVC <70% with a post bronchodilator reversibility of less than 200ml and 12% was required in all our patients. A detailed medical history was documented. We took a detailed history of dyspnoea, its duration and its severity as per mMRC grading.[9] Also a history of cough, sputum production, wheezing, chest tightness, fever, weight loss and the number of exacerbations in the previous year was noted. We then performed a detailed clinical examination, especially noting the antero-posterior / transverse chest diameter, hyper-resonance on percussion and auscultatory findings.[13] Pre and post bronchodilatory spirometry was performed in all patients as per the ATS guidelines for spirometry.[14] Persistent airflow limitation was defined by FEV1/FVC <70% and an FEV1 post bronchodilator reversibility of less 200 ml and 12%. We classified the airflow obstruction as mild- Gold 1 (FEV1>80%), moderate- Gold 2 (FEV1=50-80%), severe Gold 3 (FEV1=30-50%) and very severe Gold 5 (FEV1<30%) based on post bronchodilator FEV1.[1] We then performed an arterial blood gas analysis in all the patients. Blood was also sent for a complete haemogram. All patients then underwent an electrocardiogram (ECG) recording and a 2-Dimensional Echocardiography 92-D Echo). Pulmonary hypertension was defined as an RSVP of more than 35mmHg on 2-D Echo. High Resolution Computed Tomography (HRCT) of the chest was done in 40 patients, 10 patients refused the same. We then studied the correlation between the clinical, radiological and spirometric profiles in these patients with COPD and compared our findings with the available literature. Radiographic findings, grades of dyspnoea, hypoxia and hypercapnoea, and pulmonary hypertension were co-related with the severity of airflow obstruction.

RESULTS

A total of 50 patients were enrolled over a period of one year. Of the 50 patients enrolled, majority patients (20 patients (40%)) were between 50-59 years of age as shown in Table 1. Of the 50 patients, 33 (66%) were males while 17 (34%) were females, the male to female ratio was 1.94: 1 respectively.

Table 1: Age distribution of the study group.

Age (years)	Number of patients
40-49	10 (20%)
50-59	20 (40%)
60-69	13 (26%)
≥70	7 (14%)
Total	50

In our study, out of 50 patients, 37 (74%) had history of tobacco smoking, 6 patients (12%) had history of exposure to biomass fuel while cooking, 3 patients (6%) gave history of exposure to cotton dust and in 4 patients (8%) no particular risk was isolated (Table 2).

Table 2: Risk factor for COPD in the study group.

Risk factor	No. of patients (n=50)		Total
	Male	Female	
Tobacco smoking	30 (91%)	7 (35.4%)	37 (74%)
Air pollution (biomass)	0	6 (41.1%)	6 (12%)
Occupation (cotton mill worker)	3 (6%)	0	3 (6%)
Unknown	0	4 (23.5%)	4 (8%)
Total	33	17	

All patients presented with dyspnoea (n = 50, 100%), while cough was present in 44 patients (88%), sputum production in 34 (68%), wheezing in 29 (58%), chest tightness in 15 (30%), fever in 15 (30%) and weight loss was seen in 14 (28%) patients (Table 3).

Table 3: Clinical symptoms in the study group.

Clinical symptoms	No. of patients (n=50)	Percentage (%)
Dyspnoea on exertion	50	100%
Cough	44	88%
Sputum production	34	68%
Wheezing	29	58%
Chest tightness	15	30%
Fever	15	30%
Weight loss	14	28%

In the present study, HRCT of the chest was performed in 40 patients, of which 18 patients (45%) showed air trapping, 12 patients (30%) showed emphysema, 12 patients (30%) showed bronchial wall thickening, while only 5 patients (12.5%) had bullae and bronchial wall dilatation; and 4 patients (10%) showed atelectasis (Table 4).

Table 4: HRCT findings in our study (n=40 patients).

HRCT thorax findings	No. of patients (n=40)	Percentage
Air trapping	18	45%
Bronchial wall thickness	12	30%
Bronchial dilatation	5	12.5%
Atelectasis	4	10%
Emphysema	12	30%
Small bullae	5	12.5%
Normal	10	25%

In the current study we found that patients (70.54%) with very severe airflow obstruction had more severe hypoxia and hypercapnoea when compared to moderate airflow obstruction (Table 5).

Table 5: Relation between partial pressure of oxygen (PaO2) and partial pressure of carbon dioxide (PaCO2) in ABG and severity of airflow obstruction on spirometry.

Airflow obstruction	Average PaO2	Average PaCO2
Moderate	60.84	46.60
Severe	55.83	58.83
Very severe	46.9	59.97

The ECG showed changes of p- pulmonale with or without right ventricular hypertrophy in 21 (42%) patients (Table 6) and out of 50 patients, 27 patients (54%) had pulmonary hypertension (RVSP >35 mmHg) on 2D echocardiography while 23 patients (46%) had a normal echo test.

Table 6: ECG Findings in the study group.

ECG findings	Number of patients (n=50)
P pulmonale (RAE) only	10 (20%)
Right ventricular hypertrophy (RVH) only	4 (8%)
P pulmonale (RAE)+ RVH	11 (22%)
Normal	25 (50%)
Total	50

Table 7a: Dyspnoea severity at presentation.

Severity of dyspnoea (mMRC)	Number of patients (n=50)
Grade 0	0
Grade 1	2 (4%)
Grade 2	3 (6%)
Grade 3	4 (8%)
Grade 4	19 (38%)
Grade 5	22 (44%)

We classified dyspnoea according to modified MRC Scale, and we noted that 23 patients (44%) had grade 05 dyspnoea, 19 (38%) had grade 04 dyspnoea, 04 (8%) had grade 03 dyspnoea, 03 (6%) had grade 02 dyspnoea and 02 patients (4%) had grade 01 dyspnoea. (Table 7a) Out of the 23 patients with grade 05 dyspnoea, 17 patients (73.91%) had very severe airflow obstruction on spirometry and the remaining 6 patients (26.09%) had severe airflow obstruction. All patients (100%) with grade 01 (2 patients), grade 02 (3 patients) and grade 03 (4 patients) dyspnoea had moderate airflow obstruction on spirometry. (Table 7b) Chi square was 64.7561 and p-value was 0.00001 for the association between Grade-5 dyspnoea and very severe airflow obstruction.

In patients with documented pulmonary hypertension (27 patients) on 2D Echocardiography, 16 patients (94.1%) had very severe airflow obstruction on spirometry, 10 patients (45.45%) had severe airflow obstruction and only

1 patient (9.1%) had moderate airflow obstruction. No patient with mild airflow obstruction had pulmonary hypertension. (Table 8) There was a significant (p=0.00015) association between presence of pulmonary hypertension and very severe airflow obstruction.

Table 7b: Correlation between dyspnoea severity at presentation and severity of airflow obstruction on spirometry.

Severity of airflow obstruction	mMRC classification of dyspnoea					Total
	Grade 1	Grade 2	Grade 3	Grade 4	Grade 5	
Mild (Gold 1)	0	0	0	0	0	2
Moderate (Gold 2)	2 (100%)	3 (100%)	4 (100%)	2 (11.2%)	0	11
Severe (Gold 3)	0	0	0	16 (88.8%)	6 (26.09%)	22
Very Severe (Gold 4)	0	0	0	0	17 (73.91%)	17
Total	2	3	4	18	23	50

Chi square is 64.7561 and p- value is 0.00001 for the association between Grade-5 dyspnoea and very severe airflow obstruction.

Table 8: Correlation between pulmonary hypertension on 2D echo and severity of airflow obstruction on spirometry.

Severity of airflow obstruction	Pulmonary hypertension		Total
	Present (number of patients)	Absent (number of patients)	
Mild	0	0	0
Moderate	1 (9.1%)	10 (90.9%)	11
Severe	10 (45.45%)	12 (54.55%)	22
Very Severe	16 (94.1%)	1(5.9%)	17
Total	27	23	50

P=0.00015 for the association between presence of pulmonary hypertension and very severe airflow obstruction.

DISCUSSION

In our study, we observed that the mean age of the patients was about 58 years and the male to female ratio was 1.94: 1 respectively which is comparable to that given in literature.[15,16] Males were more affected than females due to higher prevalence of smoking and also because of increase in exposure to other risk factors in males as compared to females.9 Prevalence of COPD was more common in smokers, with smokers to non-smokers ratio of 2.83: 1.00. In our study exposure to tobacco smoke was the commonest risk factor (74%) for COPD in males followed by exposure to occupational dust (6%), whereas exposure to biomass fuel smoke (12%) was the commonest risk factor in females. The prevalence of tobacco smoking is increasing worldwide and is very strongly associated with COPD.[1] An analysis of the large US population- based National Health and Nutrition Survey III survey of almost 10,000 adults estimated that the fraction of COPD attributable to workplace exposures was 19.9%.[17] However, only 6% of our patients gave history of workplace exposure. This could be due to the fact that there is not much awareness about workplace related hazards in our country. Similarly, in females, exposure to biomass fuel is high due to the cooking practices in our villages.[18]

In the present study, 88% patients had cough, while 68% had cough with sputum production at the time of presentation which is comparable to the study by Jain NK et al.[16] About 82% patients in our study had grade 3-4 dyspnoea, as against 34.75% in study by N K Jain et al, while 18% had grade 1-2 dyspnoea.[16]

In our study HRCT of the thorax had a sensitivity of 75% in COPD. Out of 50 patients, HRCT of the thorax was performed in 40 patients, of which 25% had a normal CT. Air trapping was seen in 35% patients and bronchial dilatation was seen in 30% patients. These findings were similar to a study by Birring SS et al.[19] Radiological findings were common in patients with moderate to severe airflow obstruction, where there is more severe parenchymal damage.

In the current study we found that patients (70.54%) with very severe airflow obstruction had more severe hypoxia and hypercapnoea when compared to moderate airflow obstruction.

In the present study, ECG abnormalities were present in 50% cases as compared to a study by Niranjan et al20 where they had ECG abnormalities in 68% of their patients. We observed that 54% of our patients had pulmonary hypertension on 2 D Echo. This higher incidence could be because we had more patients with severe to very severe airflow obstruction compared to N K Jain et al.[16]

When we compared dyspnoea severity with airflow obstruction, we found a strong association between Grade- 5 dyspnoea and very severe airflow obstruction (p=0.00001 and chi square= 64.75). Out of 22 patients with Grade-5 dyspnoea, 91% had very severe airflow obstruction and the rest had severe airflow obstruction. In our study all patients with Grade 2 and 3 dyspnoea had moderate airflow obstruction, while in a comparative study only 46.1% patients with Grade 2 and 3 dyspnoea had moderate, while the rest had severe to very severe airflow obstruction.[21]

Pulmonary hypertension was more commonly seen in patients with very severe airflow obstruction, that is 94.1% patients with very severe airflow obstruction had pulmonary hypertension, which is similar to the observations made by N K Gupta et al.[22] Development of pulmonary hypertension is secondary to chronic hypoxia and hypercarbia which cause pulmonary arterial vasoconstriction. These findings are more commonly seen in association with more advanced disease with very severe airflow obstruction.

CONCLUSION

In the present study of 50 cases, COPD was seen predominantly in male patients, with a mean age of presentation between 50-59 years. Tobacco smoking was the commonest etiological factor followed by exposure to biomass fuel and occupational dust. Tobacco smoking was more common in males, while biomass fuel was more common in females. Clinical symptoms most commonly documented were dyspnoea, cough with or without expectoration, wheezing, chest tightness, fever and weight loss. There was a strong association between Grade 5 dyspnoea and very severe airflow obstruction. The common findings on HRCT were air trapping, followed by emphysema, bronchial wall thickening, bronchial dilatation and small bullae. Hypoxia, hypercarbia and pulmonary hypertension were strongly associated with very severe airflow obstruction.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Global initiative for chronic obstructive lung disease: global strategy for the diagnosis, management and prevention of chronic obstructive disease, Report; 2017.
2. Woodruff PG, Barr RG, Bleecker E. Clinical Significance of symptoms in smokers with preserved pulmonary function. N Engl J Med. 2016;374(19):1811-21.
3. Regan EA, Lynch DA, Curran- Everett D. Clinical and radiological disease in smokers with normal spirometry. JAMA Intern Med. 2015;175(9):1539-49.
4. Kohansal R, Martinez- Camblor P, Agusti A, Buist AS, Mannino DM, Soriano JB. The natural history of chronic airflow obstruction revisited: an analysis of the Framingham offspring cohort. Am J Respir Crit Care Med. 2009;180(1):3-10.
5. Eisner MD, Anthonisen N, Coultas T. An official american thoracic society public policy statement: novel risk factors and the global burden of chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 2010;182(5):693-718.
6. Paulin LM, Diette GB, Blanc PD. Occupational exposures are associated with worse morbidity in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 2015;191(5):557-65.
7. Assad NA, Balmes J, Mehta S, Cheema U, Sood A. Chronic obstructive pulmonary disease secondary to household air pollution. Semin Resp Crit Care Med. 2015;36(3):408-21.
8. Miravitlles M, Worth H, Soler Cataluna JJ. Observational study to characterize 24 hour COPD symptoms and their relationship with patient-reported outcomes: result from the ASSESS study. Respir Res. 2014;15:122.
9. Fletcher CM. Standardised questionnaire on respiratory symptoms: a statement prepared and approved by the MRC Committee on the aetiology of chronic bronchitis (MRC breathlessness score). BMJ. 1960;2:1662.
10. Sundh J, Janson C, Lisspers K, Stallberg B, Montgomery S. The dyspnoea, obstruction, smoking, exacerbation (DOSE) index is predictive of mortality in COPD. Prim Care Respir J. 2012;21(3):295-301.
11. Cho SH, Lin HC, Ghoshal AG. Respiratory disease in Asia- Pacific region: cough as the key symptom. Allergy Asthma Proc. 2016;37(2):131-40.
12. Von Haehling S, Anker SD. Cachexia as a major underestimated and unmet medical need: facts and numbers. J Cachexia Sarcopenia Muscle. 2010;1(1):1-5.
13. Cassart M, Gevenois PA, Estenne M. Rib cage dimensions in hyperinflated patients with severe chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 1996;154 (3 Pt 1)800-5.
14. Miller MR, Hankinson J, Brusasco V, Burgos F, Casaburi R, Coates A, et al. Series ATS/ERS task force: standardisation of lung function testing. Eur Respir J. 2005;26:319-38.
15. Gupta PP, Yadav R, Verma M, Agrawal D, Kumar M. Correlation between HRCT and patient characteristics in COPD. Annals Thorac Med. 2008;3(3):5-6.
16. Jain NK, Thakkar MS, Jain N, Rohan KA, Sharma M. Chronic obstructive pulmonary disease. Does gender really matter? Lung India. 2011;28(4):258-62.
17. Hnizdo E, Sullivan PA, Bang KM, Wagner G. Association between chronic obstructive pulmonary

disease and employment by industry and occupation in the US population: a study of data from the Third National Health and Nutrition Examination Survey. Am J Epidemiol. 2002;156(8):738-46.
18. Ezzati M. Indoor air pollution and health in developing countries. Lancet. 2005;366(9480):104-6.
19. Birring SS, Brightling CE, Bradding P, Entwisle JJ, Vara DD, Grigg J, et al. Clinical, radiologic and induced sputum features of chronic obstructive pulmonary disease in non- smokers. A descriptive study. Am J Respir Crit Care Med. 2002;166:1078-83.
20. Niranjan MR, Jayasheelan MR, Rashmi BK. A correlative study of spirometric parameters and ECG changes in patients with chronic obstructive pulmonary disease. Int J Biol Med Res. 2012;3(1):1322-6.
21. Chabra SK, Gupta AK, Khuma MZ. Evaluation of three scales of dyspnoea in chronic obstructive pulmonary disease. Ann Thorax Med. 2002;4(3):128-32.
22. Gupta NK, Agrawal RK, Shrivastav AB, Ved ML. Echocardiography evaluation of heart in chronic obstructive pulmonary disease patients and its correlation with severity of disease. Lung India. 2011;28(2):105-9.

Cite this article as: Kamdar DJ, Patel DK. A study of the clinical profile of 50 patients of COPD with correlation between clinical, radiological and spirometric evaluation. Int J Res Med Sci 2017;5:1802-7.