

Relationship Between Bode Index and Pulmonary Hypertension to Predict Potential Admissions in Copd

Dr. G. Veera Narayana¹, Dr. V. Purushotham², Dr. R. Radhika², Dr. B. Baby Rohini^{2*}

¹Associate Professor, Department of General Medicine, ACSR Government Medical College, Nellore, Andhra Pradesh, India

²Assistant Professor, Department of General Medicine, ACSR Government Medical College, Nellore, Andhra Pradesh, India

Corresponding author: Dr. B. Baby Rohini, Assistant Professor, Department of General Medicine, ACSR Government Medical College, Nellore, Andhra Pradesh, India

ABSTRACT

Background and objectives: To compare the BODE index's severity to the degree of pulmonary hypertension. To forecast future admissions for respiratory conditions depending on the severity of the illness as determined by the BODE index and pulmonary hypertension.

Methods: Observational research including 120 patients was conducted at Department of General Medicine, ACSR Government Medical College, Nellore, Andhra Pradesh, India from March 2023 to August 2023, after gaining approval from the institutional ethics committee.

Result: Out of the 40 patients in our research with mild pulmonary hypertension, one was admitted. 21 patients with moderate pulmonary hypertension underwent three, four, or nine admissions: once, twice, or three times. Six of the eleven patients who had significant pulmonary hypertension were hospitalised. Given that the p value is 0.001, this was statistically very significant. The rate of readmission also increased as the pulmonary hypertension did. In our investigation, we discovered that the mean Bode decreased after intervention, going from 4.2 to 3.51. However, despite interventions, the mean TRPG grew.

Conclusion: Pulmonary hypertension and COPD severity are correlated with BODE score. Patients with COPD who have a high BODE score had a lower readmission rate. With the right therapeutic intervention and rehabilitative methods, the BODE score can be raised.

Keywords: BODE index, pulmonary hypertension, COPD, PHT severity

INTRODUCTION

A deadly lung condition known as chronic obstructive pulmonary disease (COPD) causes progressively harder breathing. Despite suitable precautions, COPD prevalence and mortality are still rising as a result of ongoing exposure to risk factors, particularly smoking among women and adolescents. COPD is predicted to move up to the third most prevalent cause of death, according to GOLD estimations. Health resources must be used at great expense. The chronic obstructive pulmonary disease is used to refer to lung conditions that cause impairing of airflow and are irreversible [1,2,3]. This category mostly includes chronic bronchitis and emphysema, either separately or together. There is disagreement over whether asthma is included in this category. Nevertheless, it is typically excluded. It is classified as a restrictive lung disease even if it does contain obstructive features. The sickness of many COPD patients also includes an asthmatic component. Typically, Forced Expiratory Volume (FEV1) is the only factor used to classify the severity of COPD. Because COPD affects multiple systems, FEV1 cannot predict how these patients will fare on its own. Thus, a multidimensional grading system was created that took into account four factors: body mass index (B), the severity of dyspnea and airway obstruction, and exercise capacity (E), as determined by the six-minute walk test [4,5,6].

MATERIAL AND METHODS

After receiving consent from the institutional ethics committee, an observational study involving 120 patients was carried out at Department of General Medicine, ACSR Government Medical College, Nellore, Andhra Pradesh, India from March 2023 to August 2023. Patients with mild, moderate, and severe COPD as well as those without it provided baseline data. Ages, FEV1, the total number of admissions, and pulmonary hypertension were the parameters examined. The one-way ANOVA F-test was used to determine the significance of the difference in means between the two groups, and the Chi square test was used to determine the significance of the difference in proportions. The p-value was used to determine statistical significance when it was below 0.05.

Inclusion criteria:

1. A considerable increase in symptoms and a FEV1 that is less than 15% from the baseline value at 20 minutes after salbutamol treatment.

Exclusion criteria:

1. Past CCF or UA detection of comorbid diseases, as well as myocardial infarction within the past four months.
2. Having trouble doing the lung function and six-minute walk tests.
3. Asthma is defined as a 200 ml or rise in FEV1 after bronchodilator injection of more than 15% over the baseline value.
4. TB, bronchiectasis, or any other parenchymal lung disease patients.

Methodology

All patients who attend the medicine and chest clinic outpatient visits will be screened in accordance with the inclusion and exclusion criteria, and only those who meet the requirements and give their agreement will be enrolled. Smoking, personal, and employment histories were gathered for each individual. During the examination, height and weight were also measured. A weighing device was used to measure weight. The stadimeter was used to measure height in "mm". The formula was used to determine the body mass index (BMI).

Weight in kg / (Height in ms)² is the formula for body mass index (BMI).

They will undergo a PFT utilising a spirometer following a clinical evaluation. According to the American Thoracic Society, spirometry is carried out utilising a portable spirometer. The patient is instructed to take a deep breath after all air has been exhaled while using a spirometer. After doing this manoeuvre, the patient is instructed to quickly exhale so that their lungs are completely empty. Spirometry test results vary, although they are based on averaged, healthy population values. achieved FEV1 and FVC. FEV1 was calculated. Three readings on average were taken. The patient's history of his or her dyspnea was recorded. The patients' dyspnea was measured using the MMRC (Modified Medical Research Council) dyspnea scale [7,8,9].

RESULT**Table 1. DISTRIBUTION OF AGE AND SEX**

SEX	FREQUENCY	PERCENT
MALE	33	27.5
FEMALE	87	72.5
TOTAL	120	100.00

Table 2. PHT SEVERITY & BODE SCORE

PHT	NUMBER	MEAN	MIN	MAX.	ST.DEV.
ABSENT	49	3.0324	.00	5	0.74012
MILD	40	5.7000	3.00	7	1.40215
MOD.	21	8.135	4.00	8	1.3984
SEVERE	10	9.752	9.00	8	0.5253
TOTAL	120	5.2251	0.00	8	2.4531

$p < 0.001$ hs

The mean Bode score for the 100 patients was 3.0324, and 49 of them did not have pulmonary hypertension. According to our study, the link between the severity of pulmonary hypertension and high BODE score was statistically significant as evidenced by the p value .001. 40 patients had mild pulmonary hypertension, 10 patients had moderate PHT, and 21 patients had severe PHT (mean BODE SCORE 9.752).

Table 3. COPD Vs BODE INDEX

	No	Mean	Standard Deviation	Minimum	Maximum
MILD	45	1.698	0.621	0.00	3.00
MODERATE	45	4.587	0.921	4.00	6.00
SEVERE	30	6.702	1.088	7.00	8.00

The mean BODE score rises as the severity of COPD increases. Severe COPD patients had a mean bode score of 6.702 while those with mild COPD had a mean bode score of 1.69. Given that ($p .001$), this is statistically very significant.

Table 4. SEVERITY OF PHT & NUMBER OF ADMISSIONS

PHT	NUMBER OF ADMISSIONS				
	0.0	1.0	2.0	3.0	TOTAL
ABSENT	48	0	0	0	48
MILD	11	29	0	0	40
MODERATE	3	4	9	5	21
SEVERE	0	0	5	6	11

Out of the 40 patients with mild pulmonary hypertension in our study, one was admitted. 21 patients with moderate pulmonary hypertension had admission three times, four times, and nine times, respectively. Severe pulmonary hypertension affected 11 patients, and 6 were hospitalised. This was very significant statistically because the p value was 0.001. The rate of readmission rose along with the level of pulmonary hypertension.

Table 5. POST INTERVENTION PARAMETERS

VARIABLE	N	MEAN	STD.DEVIATION	STD.ERROR
				MEAN
P.I FEV1	120	47.600	12.4255	1.65878
P.I BODE	120	4.5310	3.65482	.44645
P.I TRPG	120	39.1200	15.5421	1.31488

Table 6. PAIRED SAMPLE TEST

	PAIRED DIFFERENCE		T	p
	MEAN	STD.DEV		
FEV1 – PIFV1	1.1548	2.4689	6.234	<0.001
BODEINDE – PIBODE	0.5468	0.31565	10.465	<0.001
TRPG - PITRPG	-1.4568	1.46948	-9.844	<0.001

In our investigation, we discovered that after intervention, the mean Bode decreased from 4.2 to 3.51. But despite interventions, the mean TRPG increased.

DISCUSSION

The ailment known as COPD (chronic obstructive pulmonary disease) causes severe morbidity and mortality. A multidimensional grading system called the BODE index performs better than the FEV1 at predicting death. When used in conjunction with the spirometric measurement of airflow (FEV1), this multistage scoring system, which evaluates symptoms, nutritional status, and exercise ability, can offer helpful prognostic information for individuals with COPD. Despite the fact that the frequency in women is steadily rising, males are far more likely than women to be diagnosed with COPD. Even in our study, 77% of the male participants had COPD, making them more prevalent. Although statistically insignificant, we discovered in our study that the prevalence of the condition rose with age above 40. These findings are consistent with research by N.S. Zhong and P.X. Ran in China. COPD affected 1 in 10 persons over the age of 40 [10,11,12].

Out of the 120 individuals, 71 developed pulmonary hypertension, 49 did not, and those who did not had a mean BODE score of 2.0233. Pulmonary hypertension affects 10 to 30% of COPD patients. According to our study, the relationship between the severity of pulmonary hypertension and high BODE score was statistically significant as evidenced by the p value .001. Six patients had severe pulmonary hypertension (mean BODE SCORE 8.1), 16 had moderate PHT (mean BODE SCORE 7.120), and 35 had light PHT. Increased lung volume, acidosis, hypoxia vasoconstriction, and secondary polycythemia all contribute to pulmonary HTN [13,14,15].

There were a total of 40 patients in the mild and moderate COPD groups, while 26 patients had severe COPD. The mean BODE scores for the mild and moderate groups were 1.72 and 4.29, respectively. The average BODE score for the group with severe COPD was 7.6154. It was determined that this was statistically significant (p 0.001) Studies by Kian-Chung *et al.* have demonstrated a strong correlation between BODE scores and severity in terms of hospitalisation and fatality. The BODE rating system may be highly useful in a developing country where there are few resources for healthcare, both for allocating resources and for future patient therapy. According to certain research, individuals with chronic obstructive pulmonary disease (COPD) can predict their survival and hospital readmissions over the course of three years using just one BODE index examination [16,17,18]. According to a study by David Hui, serial changes in the BODE index between the other time-points were not predictive of death, but a change in the BODE index score of more than one point between baseline and the six-month evaluation was slightly predictive of mortality. At 6, 12, and 24 months, however, sequential rises in the BODE index of at least one point were a reliable indicator of early readmissions to the hospital. Over time, mortality and readmissions might be predicted by a single BODE index reading. Ong *et al.* discovered that a low 6m walk test result increased hospitalisation and mortality in COPD patients in 2005. Additionally, Celli *et al.* (2004) discovered that a low 6 minute walk score increased the probability of death in COPD patients[18,19].

Adel Khattab, Khaled Wagih, and Ahmed Mohamed conducted a study. The results of utilising the BODE index as a predictor of risk of hospitalisation in COPD patients showed that risk of hospitalisation increases with rising BODE score. We discovered it in our study. Nine patients were admitted three times, with a mean BODE score of 8.2222, compared to 8 patients who were admitted twice and had a mean BODE score of 7.375. The statistical significance of this was very strong (p.001).As a result, there are more readmissions as the BODE index rises. This perfectly matched the

findings of Ong *et al.*'s 2005 study, which employed the BODE index as a predictor of hospitalisation in COPD patients and compared it to the FEV1 classification of GOLD and discovered that the BODE index was a more accurate predictor of hospitalisation than using FEV1 alone. Because hospitalisation rates were higher for people with higher BODE scores.

The response to pulmonary rehabilitation (PR) may be objectively quantified using the BODE index, and the change in the BODE index gives information regarding eventual survival, according to an observational study conducted by C.G. COTE AND B.R. CELLI over the course of a year in patients with COPD. Additionally, enrollment in a rehabilitation course is linked to a drop in hospitalisations. Rehabilitation improves dyspnea, exercise capacity, and health status but has little impact on lung function [19,20].

Exercise capacity is a predictive indicator, say Celli *et al.* Because it suggests a change in any of its components of a magnitude sufficient to affect clinical outcomes, the present authors considered one unit change in BODE as being clinically relevant. Additionally, this study showed that these patients' access to healthcare resources had improved.

Following therapies, the mean BODE SCORE decreased in our patients from 4.21 to 3.51, a significant decrease in just six months. Despite treatment, the FEV1 levels decreased, indicating a decline in lung function. The slight increase in TRPG levels indicates rising PHT. Despite the fact that COPD is a progressive condition, the research mentioned above have decisively shown that it is possible to determine post-treatment improvement. Despite conflicting data about the advancement of the primary disease issue per se, it has been demonstrated that the BODE score, a composite index of COPD severity, significantly decreases during treatment [20,21].

CONCLUSION

The severity of pulmonary hypertension and COPD are correlated with BODE score. The number of readmissions for COPD patients can be predicted by the BODE score. By using appropriate and timely therapeutic intervention and rehabilitative techniques, BODE score can be improved.

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REFERENCE:

1. Burden of Obstructive Lung Disease (BOLD) Project in South China. N.S. Zhong, P.X. Ran, J.C. Lu, S.M. Liu, J.P. Zheng, W.M. Vollmer, A.S. Buist. *Am J Respir Crit Care Med* 2004 169(7): A603.
2. National Center for Health Statistics. Current estimates of the National Health Interview survey, United States, 1995. Washington DC: Department of Health and Human Services, Public Health Service, Vital and Health Statistics; 1995. Publication No. 96-1527.
3. National Heart, Lung, and Blood Institute. Morbidity and mortality: chartbook on cardiovascular, lung, and blood diseases. Bethesda MD: US Department of Health and Human Services. Public Health Service, NIH Available from URL.
4. Soriano JR, Maier WC, Egger P, Visick G, Thakrar B, Sykes J, *et al.* Recent trends in physician diagnosed COPD in women and men in the UK. *Thorax* 2000; 55: 789-94.
5. Murray CJL, Lopez AD. Evidence-based health policy – lessons from the Global Burden of Disease Study. *Science* 1996; 274: 740-3.
6. Khan MM, Tandon SN, Khan MT, Pandey US, Idris MZ. A comparative study of effects of cigarette and bidi smoking on respiratory function tests. *J Environ Biol* 2002;23:89-93.
7. Jaakkola MS, Jaakkola JJ. Effects of environmental tobacco smoke on the respiratory health of adults. *Scand J Work Environ Health* 2002;28 Suppl 2:52-70.
8. Smith KR. National burden of disease in India from domestic air pollution. *Proc Natl Acad Sci* 2000;24:13286-13293.
9. Pandey MR. Domestic smoke pollution and chronic bronchitis in a rural community of the Hill Region of Nepal. *Thorax* 1984;39:337-339.
10. Behera D, Jindal SK. Respiratory symptoms in Indian women using domestic cooking fuels. *Chest* 1991;100:385-388.
11. Perez-Padilla R, Regalado U, Vedal S, *et al.* Exposure to biomass smoke and chronic airway disease in Mexican women. *Am J Respir Crit Care Med* 1996;154:701-706.
12. Becklake MR. Occupational exposures: evidence for a causal association with chronic obstructive pulmonary disease. *Am Rev Respir Dis.* 1989 Sep;140(3 Pt 2):S85-S91.
13. Oxman AD, Muir DC, Shannon HS, Stock SR, Hnizdo E, Lange HJ. Occupational dust exposure and chronic obstructive pulmonary disease. A systematic overview of the evidence. *Am Rev Respir Dis* 1993;148:38-48.
14. Sunyer J. Urban air pollution and chronic obstructive pulmonary disease: a review. *Eur Respir J* 2001;17:1024-1033.

15. Karakatsani A, Andreadaki S, Katsouyanni K, Dimitroulis I, Trichopoulos D, Benetou V, *et al.* Air pollution in relation to manifestations of chronic pulmonary disease: a nested case-control study in Athens, Greece. *Eur J Epidemiol* 2003;18:45-53.
16. Mannino DM, Homa DM, Akinbami LJ, Ford ES, Redd SC. Chronic obstructive pulmonary disease surveillance– United States, 1971-2000. *MMWR Surveill Summ* 2002;51(6):1-16.
17. Xu X, Weiss ST, Rijcken B, Schouten JP. Smoking, changes in smoking habits, and rate of decline in FEV1: new insight into gender differences. *Eur Respir J* 1994;7(6):1056-61.
18. Anthonisen NR, Connett JE, Kiley JP, Altose MD, Bailey WC, Buist AS, *et al.* Effects of smoking intervention and the use of an inhaled anticholinergic bronchodilator on the rate of decline of FEV1. The Lung Health Study. *JAMA* 1994;272(19):1497-505.
19. Silverman EK, Weiss ST, Drazen JM, Chapman HA, Carey V, Campbell EJ, *et al.* Gender-related differences in severe, early-onset chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 2000;162(6): 2152-8.
20. Siafakas NM, Vermeire P, Pride NB, *et al.* Optimal assessment and management of chronic obstructive pulmonary disease (COPD). The European Respiratory Society Task Force. *Eur Respir J* 1995; 8:1398– 1420.
21. Pauwels RA, Buist AS, Calverley PM, *et al.* Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. NHLBI/WHO Global Initiative for Chronic Obstructive Lung Disease (GOLD) workshop summary. *Am J Respir Crit Care Med* 2001; 163: 1256–1276.