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Research Article

A Study On The Correlation Between Clinical Features, Spirometry And Abg In Acute Exacerbation Of Obstructive Lung Disease

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ABSTRACT

Background: Limitation of exhaling capacity of air due to narrowing or damage of conducting tract or lungs is known as obstructive lung disease. Obstructive lung disease may consist of Bronchial asthma, cystic fibrosis, COPD and bronchiectasis marked by exacerbation and worsening of clinical presentation. Quantitization of impairment can be measured by bedside spiromentry measuring the FEV1 and FVC whereas exchange through alveoli may be measured by arterial blood gas analysis. The study depolys the tools readily available namely spirometry and ABG to correlate clinical features of 100 patients presenting with acute exacerbation of obstructive lung disease in a population setting of rural south india. The present study also aims to establish a predicting confidence of FEV1 value to ABG value changes and its use as a predictive marker for respiratory failure.

Methods: 100 patients presenting with acute exacerbation of obstructive lung disease in the department of general medicine VMKV MCH were subjected to Spirometry and ABG on admission and on the date of discharge.

Results: PH <7.20 and spirometry showing pCO2 of more than 60 with paO2 less than 60 with FeV1 of less than 68% were found to be major factors associated with respiratory failure.

Conclusion: there is a significant correlation between FEV1 and pCO2 with clinical feature of acute exacerbation of obstructive lung disease. A Combination of spirometry with ABG increases the overall predicting acuracy of respiratory failure.

Key words: ABG, Obstructive lung disease, FEV1

INTRODUCTION:

Obstructive lung disorder of which chronic obstructive lung disease (COPD) is the fourth highest mortality causing disease and twelveth highest morbidity causing disorder. 15% of elderly are affected by this disease and ranks 6th on disase causing death world wide[3,4].

Episodic event of breathlessness, excessive sputum production, cough, dyspnea labelled as “exacerbations” plague the chronicity and denotes the progressive nature of the disease. Having a negative impact on the day to day life as well as quality of life these exacerbations can result in recuurent hospital admission and death[1].

Mortality as high as 20.3% have been noted in such casees.

Characterized by narrowing of expiratory airflow or its limitation; it can be measured by bedside spirometry. ABG can note the fault in exchange. Other test for evaluation include Xray chest, sputum analysis other higher radiological investigation. This study aims to establish a correlation between clinical features and values obtained from spirometry and ABG with a intention of establishing a positive predicting correlation for respiratory failure in patient of COPD.

Materials And Methods:

Study design : Prospective Observation study
Duration of study :1 year
Study Sample: 100 cases of acute exacerbation of obstructive lung disease presenting to VMKV MCH Inclusion criteria: patient with acute exacerbation with age group of 18-85 years.

Exclusion criteria :

1. Age <18, >85
2. Stable Obstructive lung disease.
3. Patient with restrictive lung disease or tuberculosis
4. COVID -19

Methodology:

100 patients underwent the current study who presented with acute exacerbation of obstructive lung disease in General medicine department of VMKV meeting the inclusion and exlusion criteria. Ethical committee clearance was obtained and with due consent patients were enrolled. Clinical History and examination was performed in detail and the findings were entered in the proforma.
On admission Spiromentry and ABG was done whose results were included in the proforma as well.
Laboratory methods:

Result:

Gender distribution:

Gender	Percentage
Male	80
female	20
total	100

The above table shows the result for age distribution showing 80% male and 20% female. Age distribution:

Age	mild	moderate	total	percentage
71-80 years	0	2	2	2
61-70 years	10	5	15	15
51-60 years	25	5	40	40
41-50 years	5	15	30	30
31-40 years	2	8	10	10
21-30 years	2	1	3	3

Age distribution shows more demographic towards 51-60 years of adult age in which the mean age was 58 years

Gold criteria for COPD severity

	Mild/ stage 1	Moderate/ stage 2	Severe/ stage 3	Very stage severe/ 4
Male (80)	44	36	0	0
Female (20)	19	1	0	0
Total(100)	63	37	0	0

Occupation:
Occupation of the participants obtained on detailed history shows most common being day to day labour followed by farmer.

Place of residence:
All the participants of the current study were resident of chinnaseeragapadi salem.

Smoking:
Analysed data on history showed smoking being the major risk factor associated with 80.9% of mild disease group (N=51) and 97.29% of moderate (N=36).
Mean BMI of the participant were 22.9 with a standard deviation of 2.1

Clinical features:

Clinical features	N	Y
Exertional dyspnea	96	4
hemoptysis	100	0
cough	8	92
Increased sputum	6	94
fever	94	6
wheeze	0	100
Reduced exercise tolerance	0	100
fatigue	84	16
chronic	0	100

Most common presentation of patient was reduced exercise tolerance followed by acute increase in sputum production associated with wheeze. Haemoptysis was the least frequently obtained complain.

Classification of chronic onstructive pulmonary disease according to gold criteria on admission

Characteristics		mild	moderate	total	percent	P value
Exertional dyspnea	N	54	42	96	96	0.691
	Y	2	2	4	4	
Exercise intolerance		56	44	100	100	
wheeze		56	44	100	100	
Chronic cough	N	4	4	8	8	0.598
	Y	52	40	92	92	
fatigue	N	46	38	84	84	0.498
	Y	10	6	16	16	
fever	N	54	40	94	94	0.409
	Y	2	4	6	6	
smoking	N	46	36	82	82	0.629
	Y	10	8	18	18	
Cad	N	52	42	94	94	0.591
	Y	4	2	6	6	

Htn	N	50	42	92	92	0.402
	Y	6	2	8	8	
DM	N	46	42	88	88	0.160
	Y	10	2	12	12	

Spirometry and ABG association on admission:

ABG	Respiratory failure	N	mean	95% confidence level		Standard deviation	T test	P value
				lower	upper			
pH	yes	20	7.1290	-.12368	.00218	0.08698	-1.941	.058
	no	80	7.1898			0.08888		
pO2	yes	20	91.50	-4.153	8.253	8.489	.729	.482
	no	40	89.45			5.311		
pCO2	yes	20	58.60	2.500	8.300	5.358	3.744	.000
	no	40	53.20			3.722		
HCO3	yes	20	15.80	-1.183	.833	.919	-.349	.729
	no	40	15.98			1.510		

The above table clearly shows that the mean pCO2 in patients with respiratory collapse/failure was 58.6 and in other participants was 53.2. significant association was obtained on analysis of ABG and respiratory failure with only pCO2 as the variable of study.

Spirometry	Respiratory failure	N	Mean	Standard deviation	95% confidence interval		T test	P value
					Lower	Upper		
FEV1	yes	20	68.10	8.439	-5.781	5.931	.026	.980
	no	80	68.03	8.192				
FVC	yes	20	81.20	5.287	-4.631	3.581	-.257	.798
	no	80	81.73	5.883				
FEV1/FVC	yes	20	83.60	10.885	-6.689	7.830	.159	.874
	no	80	83.03	10.057				

The above table depicts that mean FEV1 in participants was 68% and FVC was 81% with mean FEV1/FVC was 83%. There was no siggnificant association between spirometry and ABG.

Disscussion:

Gender - 20% of the study population were female. This is very low as compared to western data. Steer et al[27] study showed 42-53%. A factor for such less demographic might be due to less prevalence of smoking in females of india as well as reduced medical heath care delivery to women. COPD prevalence is increasing worldwide in female population and in India it can be due to passive smoking, biomass fuel and passive smoking.

Age- Average age when comapred with Emerman et al which was 64 is less in the current study being 58 with mean BMI being 23.8.

Smoking- A dose-response relationship was found in the current study which is a major etiology found in all study conducted previously. Smoking is a major risk factor for COPD and its association is well established.
Occupation: compounding with smoking farming was the major occupation found with the current study. Farmers being exposed to noxious agents are already at risk but smoking increases the incidence for COPD[4].
Chest Xray- the representation of consolidation in chest xray was less as compared to sanders et all[5] and lieberman et all[28].
Spirometry- Swanney et al[6] documented that FEV1/FVC is a important scale for defining COPD. Same was established in the current study. 0.5-0.69 range was seen in the study for FEV1/FVC. This proved significant relationship for severity of COPD. A ph of 7.43 and pCO2 of >45 and pO2<60 was seen in initial ABG on admission showing no significant correlation. Moderate correlation as seen in Emerman et al [8] and gupta et al was seen in the current study.
Association of respiratory failure and pCO2>45 and pO2<50 was seen in murray et al[7] was founded which is seen in our study as well.Emerman et al concluded that ABG was a poor indicator for prediction of respiratory failure.Khilani et al[9] , Hoo et al[10]. also identified a pH<7.25 and <7.2 to be significant predictor for need of intubation for the ventilation perfusion missmatch in such cases.Kumar et al[11] showed a significant need for intubation with pCO2 >60mmhg.Gompertz et al[12] and Anja et al[13] showed streptococcus being most common cause of exacerbation in infection followed by H. influenza. Madhvi et al[14] showed significant association of moraxella with exacerbation. In current study klebsiella pneumonia was found to be the major etiology followed by pneumococcus follwed by staphylococcus. Our study shows gram negative organism being more prevalent etiologically than other studies showing gram positive predominance.

Conclusion.

- With age the severity of acute exacerbation also increses.[16]
- Male predominanceis seen.[17]
- Smoking is the major etiological factor seen.[18]
- Occupation compounds the major etiological factor.[19]
- The average BMI is 24 at presentation.[20]
- Raised pCO2 and reduced pO2 is on admission is seen with raised incidence of failure of respiratory system.[21]
- pH 7.11-7.20 were at risk of developing repiratory failure[22]. ☐ pCO2>60 and pO2<60 is majorly associated with failure.[23]
- FEV1<68% is also associated with the same.[24]
- There is no correlation between pO2 and FEV1 in prediction and severity of exacerbation in COPD.[25]
- Weak correlation of FEV1 anf pCO2 in prediction of aim is observed.[26]
- ABG should be done at FEV1<68% is proposed to prediction and gauge the severity of COPD.[27]
- We conclude that patient with pCO2<45 and FEV1>45 may be discharged if asymptomatic following complition of treatment.[28]
- No significant change in patients post discharge with repect to variables in the study were noted.[31]
- Gram nehative klebsiella was the major etiological factor seen in acute exacerbation of COPD.[29]

References:

1. . Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary isease GOLD Executive Summary Jorgen Vestbo 1, 2, Suzanne S. Hurd3, Alvar G. Agusti '4, Paul W. Jones5, Claus Vogelmeier6,Antonio Anzueto7, Peter J. Barnes8, Leonardo M. Fabbri9, Fernando J. Martinez10, Masaharu Nishimurall, Robert A. Stockley12, Don D. Sin13, and Roberto Rodriguez- Roisin4. Am Respir Crit Care Med Vol 187, Iss. 4, pp 347-365, Feb 15, 2013. Copyright a 2013 by the American Thoracic Society Originally Published inPress as DOI: 10.1164/rcm.201204-0596PP on August 9, 2012
2. National Heart, Lung, and Blood Institute. Guidelines for the Diagnosis and Management of Asthma (EPR-3). 2007. http://www.nhlbi.nih.gov/guidelines/ asthma/index.htm. Accessed July 16, 2015.
3. Buist AS, Mc Burnie MA, Vollmer WM, et al. International variation in the prevalence of COPD (the BOLD Study): a population-based prevalence study. Lancet. 2007;370:741-750.
4. Vollmer WM, Gislason T, Burney P, et al. Comparison of spirometry criteria for the diagnosis of COPD: results from the BOLD study. Eur Respir J. 2009;34:588-597
5. Aggarwal AN, Chaudhry K, Chhabra SK, D Souza GA,Gupta D, Jindal SK, Katiyar SK, Kumar R, Shah B,Vijayan VK. Prevalence and risk factors for bronchial asthma in Indian adults: a multicentre study. Indian Journal of Chest Diseases and Allied Sciences. 2006 Jan 18;48(1):13.
6. Svanes C, Sunyer J, Plana E, et al. Early life origins of chronic obstructive pulmonary disease. Thorax 2009; [Epub ahead of print DOI: 10.1136/thx.2008.112136]
7. Moffatt MF, Kabesch M, Liang L, et al. Genetic variants regulating ORML3 expression contribute to the risk of childhood asthma. Nature. 2007;448:470-3

8. Bunyavanich S, Schadt EE. Systems biology of asthmaand allergic diseases: A multiscale approach. J Allergy Clin Immunol. 2015;135:31-42
9. Tsang KW, Lam SK, Lam WK, et al. High seroprevalence of Helicobacter pylori in active bronchiectasis. Am J Respir Crit Care Med. 1998 Oct.158(4):1047-51.
10. Gadek JE, Fells GA, Crystal RG. Cigarette smokinginduces functional antiprotease deficiency in the lower respiratory tract of humans. Science. 1979;206:1315- 1316.
11. National Heart, Lung, and Blood Institute. Guidelinesfor the Diagnosis and Management of Asthma (EPR-3).2007. http://www.nhlbi.nih.gov/guidelines/asthma/index.htm. Accessed July 16, 2015.
12. Papadopoulos NG, Arakawa H, Carlsen KH, et al. international consensus on (ICON) pediatric asthma. Allergy. 2012;67:976-997.
13. Holgate ST. Pathogenesis of asthma. Clin Exp Allergy. 2008 Jun;38(6):872-97. doi: 10.1111/j.1365-2222.2008.02971.x. PMID: 18498538.
14. Knox AJ. How prevalent is aspirin induced asthma? Thorax. 2002;57:565-566. Wenzel S. Severe/fatal asthma. Chest. 2003;123:405S-410S.
15. Robert F, William W. Asthma. J Allergy and Clinical Immunology. 2003;111(1):S49-S61. Busse WW,Lemanske RF Jr. Asthma. N Engl J Med. 2001;344:350-62.
16. Relationship between asthma and rhinitis: Epidemiological, Pathophysiological And therapeutic aspects-C. Bergeronetal. Allergy, Asthma and Clinical Immunology. 2005;1:81-87.
17. Wenzel S. Severe/fatal asthma. Chest. 2003;123:405S-410S.
18. Sears MR. Epidemiology of asthma exacerbations. Journal of Allergy and Clinical Immunology. 2008 Oct1;122(4):662-8.
19. Vignola AM, J. Bousquet J, Tissue remodeling as a feature of Persistentasthma. J Allergy Clin Immunology. 2000:105:1041-33.
20. Thomas AJ, Apiyasawat S, Spodick DH. Electrocardiographic detection of emphysema. Am J Cardiol. 2011;107:1090-2.
21. Ulrik C, Peripheral eosinophil counts as a marker of disease activity in intrinsic and Extrinsic asthma. Clin Exp Allergy. 1995:25(9):820-7. N Eng J Med. 1975:292(22);1152-5.
22. Horn B, Robin ED, Theodore J, Van Kessel A, Total eosinophil counts in management of Bronchial asthma.
23. Platts Mills TA. The role of immunoglobulin E in allergy and asthma. Am J Respir Crit Care Med. 2006:164:Sl-S5.
24. Borish L, Chipps B, Deniz Y, et al. Total serum IgE in a large cohort of patient with severe or difficult to treat asthma. Ann allergy Asthma Immunology. 2005;95:247-53
25. Sanders C. The radiographic diagnosis of emphysema. Radiol Clin North America. 1991;29:1019.
26. Snider GL Nosology for our day. Its application to COPD. Am J Respir. Crit. Care Med. 2003;167:678-683.
27. Clinical approach to patients with COPD and Cordiovascular disease; Stephen I, Rennard Am Thorac Soc. 2005;2:94-100.
28. COPD - Problems in diagnosis and measurement. Eur. Respir. J. 2003;21:Suppl. 41.
29. Lieberman D, Lieberman D, Gelfer Y, et al. Pneumonic vs nonpneumonic acute exacerbations of COPD. Chest 2002;122:1264e70.
30. Steer J, Norman EM, Afolabi OA, et al. Dyspnoea severity and pneumonia as predictors of in-hospital mortality and early readmission in acute exacerbations of COPD. Thorax. 2012;67:11721.
31. Costello R, Deegan P, Fitzpatrick M, McNicholas WT. Reversible hypercapnia in chronic obstructive pulmonary disease: a distinct pattern of respiratory failure with a favourable prognosis. Am J Med. 1997;102:239-244.