

Association Between Amount Of Smoking With Chronic Cough And Sputum Production

M Movahed, N Milne

Citation

M Movahed, N Milne. *Association Between Amount Of Smoking With Chronic Cough And Sputum Production*. The Internet Journal of Pulmonary Medicine. 2006 Volume 7 Number 1.

Abstract

Background: Smoking is a major cause of respiratory disease. We evaluated the association between the amount of chronic smoking with chronic cough and sputum production as a surrogate of pulmonary abnormalities

Methods: 139 patients who underwent left ventricular ejection fraction measurement for clinical reasons, were evaluated for the presence of chronic cough and sputum production with reference to the length and amount of smoking.

Results: The length and amount of smoking increased the risk of sputum production (smoking less than 10 pack years (9 of 48 18.8% vs smoking more than 10 pack years 38 of 108, 35.2%, OR: 1.3 CI 1.03-1.5, p=0.03). The length of smoking over 50 pack years vs less than 5 pack years was also significantly associated with chronic cough (24 of 31, 77.4% vs 22 of 52, 42.3%, OR: 4.7 CI: 1.7-12.7, p =0.002). Furthermore the mean length of smoking was significantly longer in patients with chronic cough (34.9 ± 38.2 vs 49.1 ± 40.2, p=0.03) or increased sputum production (34.6 ± 39 vs 56.0 ± 40.4, p=0.002).

Conclusion: Increasing in length and amount of smoking is associated with increase risk for chronic cough and sputum production consistent with dose dependent negative effect of chronic smoking.

BACKGROUND

Smoking is a major cause of cardiovascular and respiratory disease. It is not known when early pulmonary abnormalities occur in the chronic smoker. Many epidemiological studies have found that cigarette smoking is by far the most important risk factor for chronic obstructive lung disease (COPD).⁽¹⁾ Furthermore, cigarette smokers have more respiratory symptoms and lung function abnormalities as early signs of COPD.^(2, 3) It is also known that that total pack-years of smoking are predictive of COPD mortality.⁽⁴⁾ Although not all smokers develop clinically significant COPD, per epidemiological study, if they live long and smoke long enough, they will develop airway limitation.⁽⁵⁾ However, the onset of respiratory symptoms in relation to the amount of smoking is not well studied. The goal of this study was to evaluate association between the timing and amount of chronic smoking with chronic cough and sputum production as a measure of pulmonary abnormalities in chronic smoker.

METHODS

Randomly selected 139 patients at the Long Beach Veterans Administration (VA) Medical Center in California, who underwent left ventricular function assessment for clinical indications were evaluated for our study. Medical histories including history of chronic sputum production, cough, history of second hand smoking and the severity and length of smoking were documented using chart reviews and history from the patients. Using Fisher's Exact and Chi square Test, we evaluated any association between the amount and length of smoking with cough and chronic sputum production.

Results: Male gender was predominant gender in a VA population (91%). The length and amount of smoking increased the risk of sputum production (smoking less than 10 pack years (9 of 48 18.8% vs smoking more than 10 pack years 38 of 108, 35.2%, OR: 1.3 CI 1.03-1.5, p=0.03). The length of smoking over 50 pack years vs. less than 5 pack years was also significantly associated with symptoms of chronic cough (24 of 31, 77.4% vs. 22 of 52, 42.3, OR: 4.7

CI: 1.7-12.7, $p=0.002$) Using a cut-off point of less or more than 10 pack years of smoking, increased prevalence of chronic cough showed a trend toward smoking of more than 10 pack years. (42 of 105, 40% vs 12 of 48, 25%, OR: 2.0 CI 0.9-4.3, $p=0.07$). Furthermore the mean length of smoking was significantly longer in patients with chronic cough ($34.9 \text{ y} \pm 38.2 \text{ y}$ vs 49.1 ± 40.2 , $p=0.03$) or increased sputum production ($34.6 \text{ y} \pm 39$ vs $56.0 \text{ y} \pm 40.4$, $p=0.002$).

DISCUSSION

Our results are consistent with the increased risk of lung toxicity of chronic smoking with the time and amount of smoking. Increase in sputum production and cough in patients with smoking or COPD has been documented.^(6, 7) Tobacco use is by far the most important risk factor for respiratory symptoms and cough^(8,9,10) and active smoking has been found to be the most important single factor leading to development of COPD.^(9, 11) In our study pulmonary symptoms appears to increase in frequency once 10 years-pack history is reached. As many patients with early COPD are asymptomatic,⁽¹²⁾ the damaging effect of smoking on the lung function must occur earlier than 10 years-pack smoking and is very difficult to predict. Based on our study, the early occurrence of pulmonary dysfunction after 10 years-pack history of smoking, suggests that early smoking cessation is required in order to decrease the prevalence of COPD. Our findings are consistent with Lindberg et al.⁽³⁾ who found of high cumulative incidence of COPD (13,5%) after 10 years of smoking. This emphasizes the importance of early smoking cessation in the reduction of incidence of COPD.

LIMITATION

Men were the predominant gender in our study limiting our results to men. This study was not a prospective randomized trial. We used chronic cough and sputum production as a measure of abnormal lung function which is not specific limiting our study. We did not have baseline occupational history or any data about severity of symptoms limiting our results.

CONCLUSION

Increase in length and amount of smoking are associated with increased risk for chronic cough and sputum production consistent with dose dependent negative effect of chronic

smoking.

CORRESPONDENCE TO

M. Reza Movahed, MD, PhD, FACP, FCCP Associate Professor of Medicine Director of Coronary Care Unit University of Arizona Sarver Heart Center Department of Medicine, Division of Cardiology 1501 North Campbell Ave. Tucson, AZ, 85724 Tel: (520)-626-2000 E mails: rmovahed@email.arizona.edu rmova@aol.com

References

1. 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.
2. Burrows B, Niden AH, Barclay WR, Kasik JE. Chronic Obstructive Lung Disease. Ii. Relationship of Clinical and Physiologic Findings to the Severity of Airways Obstruction. *Am Rev Respir Dis* 1965;91:665-78.
3. Lindberg A, Jonsson AC, Ronmark E, Lundgren R, Larsson LG, Lundback B. Ten-year cumulative incidence of COPD and risk factors for incident disease in a symptomatic cohort. *Chest* 2005;127(5):1544-52.
4. de Marco R, Accordini S, Cerveri I, et al. An international survey of chronic obstructive pulmonary disease in young adults according to GOLD stages. *Thorax* 2004;59(2):120-5.
5. Lundback B, Lindberg A, Lindstrom M, et al. Not 15 but 50% of smokers develop COPD?--Report from the Obstructive Lung Disease in Northern Sweden Studies. *Respir Med* 2003;97(2):115-22.
6. Lindstrom M, Kotaniemi J, Jonsson E, Lundback B. Smoking, respiratory symptoms, and diseases : a comparative study between northern Sweden and northern Finland: report from the FinEsS study. *Chest* 2001;119(3):852-61.
7. Gulsvik A. Prevalence and manifestations of obstructive lung disease in the city of Oslo. *Scand J Respir Dis* 1979;60(5):286-96.
8. Huhti E. Prevalence of respiratory symptoms, chronic bronchitis and pulmonary emphysema in a Finnish rural population. Field survey of age group 40-64 in the Harjavalta area. *Acta Tuberc Pneumol Scand* 1965;Suppl 61:1-111.
9. Lundback B, Nystrom L, Rosenhall L, Stjernberg N. Obstructive lung disease in northern Sweden: respiratory symptoms assessed in a postal survey. *Eur Respir J* 1991;4(3):257-66.
10. Senior RM, Anthonisen NR. Chronic obstructive pulmonary disease (COPD). *Am J Respir Crit Care Med* 1998;157(4 Pt 2):S139-47.
11. Snider GL. Chronic obstructive pulmonary disease: risk factors, pathophysiology and pathogenesis. *Annu Rev Med* 1989;40:411-29.
12. Pauwels RA, Rabe KF. Burden and clinical features of chronic obstructive pulmonary disease (COPD). *Lancet* 2004;364(9434):613-20.

Author Information

Mohammad-Reza Movahed, M.D., Ph.D.

Department of Medicine, Section of Cardiology, University of Arizona Sarver Heart Center

Norah Milne, M.D.

Department of Radiology, Division of Nuclear Medicine, University of California, Irvine Medical Center