AJMS
(A US National Library of Medicine enlisted journal)
Al Ameen J Med Sci (2011)4(4):391-395
ISSN 0974-1143
CODEN: AAJMBG

SHORT COMMUNICATION

An Altered Pulmonary Function – A Cause or Consequence of Gastro Esophageal Reflux Disease (GERD)

H. Manjunath[1], D. Venkatesh[2*], Umesh Jalihal[3] and M. Prashanth Kumar[4]

[1]Department of Physiology, Navodaya Medical College, Raichur, Karnataka, India [2]Department of Physiology, M S Ramaiah Medical College, Bangalore, Karnataka, India [3]Department of Gastroenterology, M S Ramaiah Medical College, Bangalore, Karnataka, India and [4]Department of Physiology, MVJ Medical College, Bangalore, Karnataka India

Abstract: *Objective:* To evaluate the pulmonary function in Gastroesophageal reflux disease (GERD) patients without respiratory symptoms. *Background:* GERD is reported to adversely impact the pulmonary functions. GERD is associated with symptomatic asthma and chronic cough. Pulmonary functions are studied in symptomatic asthma patients with GERD. Treatment of GERD improves the respiratory symptoms and decreases the medication needed for the management of asthma. This study was undertaken to evaluate the pulmonary functions in GERD patients without respiratory symptoms. *Methods:* A cross sectional study was conducted in clinically diagnosed GERD cases (18 males and 15 females), confirmed by endoscopic examination without respiratory symptoms. Age, gender and anthropometrically matched healthy subjects served as controls. Pulmonary functions (FVC, FEV_1, FEV_1/FVC ratio and PEFR) were recorded using a computerized spirometer (Spirobank G) in sitting position. Percentage of predicted values was expressed as mean ± SD and was used for comparison. *Results:* There was a statistically significant reduction in FVC, FEV_1 and PEFR in GERD patients without respiratory symptoms compared to controls ($p < 0.05$). *Conclusion:* It can be concluded that subjects suffering from GERD without respiratory symptoms also have an altered pulmonary function showing predominantly obstructive type with mild to moderate restrictive airway changes.

Key words: Gastroesophageal reflux disorder, asthma, esophagitis, bronchospasm

Introduction

Gastro esophageal reflux disease (GERD) is caused by reflux of gastric contents into the esophagus. The diagnosis is made if the patient complains of heartburn, regurgitation and endoscopy shows signs of esophagitis. It produces esophageal and extraesophageal manifestations [1]. The commonest extraesophageal disorder involves chronic cough and asthma like features. However it is difficult to establish the exact cause and consequence relationship between reflux and respiratory symptoms. Lack of clarity in understanding this relationship can put the treating physician in a dilemma and can pose a therapeutic challenge. There have been efforts in the recent past to establish a link between GERD and bronchial asthma. It has been observed that these two conditions co-exist and reflux could trigger or aggravate asthma [2]. Esophageal acidification altered the pulmonary function and antireflux therapy has reduced the requirement of drugs for management of asthma by 75 % [3-4].

Esophagitis was detected in about 40 % of asthmatic subjects [5]. Further, pH studies established GERD in about 25-60% of asthmatics without reflux symptoms [6-7]. The prevalence of GERD in asthmatics has been established by endoscopy and ambulatory pH monitoring [2, 8]. Young adults with nocturnal reflux symptoms had higher prevalence of asthma as compared to patients without reflux [9]. GERD is considered to be one of the most common causes for chronic cough lasting for more than 8 weeks [10-11]. In the earlier studies efforts have been made to establish the relationship between GERD and asthma, chronic cough. Few of the earlier studies have proposed mechanisms to explain the development of asthma and chronic cough in cases of GERD where as some of the studies have explained mechanisms for manifestation of GERD in cases of bronchial asthma. The present study is undertaken to evaluate the effect of gastroesophageal reflux on pulmonary functions in GERD patients without respiratory symptoms.

Material and Method

Institutional ethical committee for human research gave the clearance for the study [12]. The subjects were explained the study protocol, possible risks involved in participation, extent of involvement and the right to withdraw from the study. An informed written consent was taken from all the subjects. Thirty three patients attending department of gastroenterology at M.S. Ramaiah hospitals were recruited for the study. Age, gender and anthropometrically matched 33 subjects attending the master health checkup were recruited as controls. Smokers, subjects/patients with ischemic heart disease, respiratory disorder and diabetes mellitus were excluded from the study. GERD was diagnosed based on the presenting complaints, GERD symptoms questionnaire, and confirmed by endoscopic examination. GERD patients taking medications like antacids, H_2 blockers or proton pump inhibitors were excluded from the study. The subjects underwent upper GI endoscopy for evaluation of reflux Esophagitis and were graded according to Los Angeles classification [13]. Complete blood counts, random blood sugar, chest X ray and ECG were done to all the recruited subjects to rule out the existence of other medical problems. Spirometry was performed under standard conditions in sitting position in air conditioned room under controlled temperature and humidity. Pulmonary function tests were performed between 9 am and 1 pm in order to eliminate circadian influences. The subjects rested for duration of 15 minutes, performed spirometry after detailed instruction and adequate practice sessions. Respmed Spirobank G, MIR SRL, Italy was used for evaluation of pulmonary functions. Each subject performed three trials, with at least two reproducible trials according to the recommendations of American thoracic society [14]. The values were considered reproducible when the differences between the two highest recorded values were not more than 5%. The best values of FVC, FEV_1, FEV_1 % and PEF were used for comparison and evaluation. BMI was calculated by measuring weight in kg and height in meters. Waist circumference was measured at the level midway between lower rib margin and iliac crest. Hip circumference was measured as the maximal circumference over the buttocks. Waist-hip ratio was calculated by using the measurements.

Statistical analysis: Descriptive statistical analysis has been performed in the present study. Results on continuous measurement were expressed as mean ± SD and results on categorical measurements were presented in numbers (%). Significance was assessed at 5% level of significance. Two tailed independent student 't' test was used to find the significance of study parameters between two groups, two tailed dependent student 't' test was used to find the significance of study parameters between predicted and recorded values. Mann Whitney U test was used to find the significance of parameters on non-parameters. Wilcoxon's singed rank test was used to find the significance within each group between the observed and recorded values.

Result

A cross sectional study of 33 GERD patients and 33 controls consisting of 18 males and 15 females in each group was undertaken. The age of patients and the controls subjects were 42.0 ± 11.32 years and 42.4 ± 11.18 years respectively. There was no significant difference between the BMI, waist-hip ratio between GERD patients and control subjects in both males and females (Table 1).

Table-1: Comparison of study characteristics between two groups

Study characteristics	Cases	Controls	p value
Age in years	42.00 ± 11.32	42.45 ± 11.18	0.870
Gender (M:F)	18:15	18:15	1.000
BMI (kg/m^2) (Males)	26.68 ± 5.56	25.89 ± 4.61	0.419
BMI (kg/m^2) (Females)	27.27 ± 4.4	27.64 ± 4.26	0.814
Waist /Hip ratio (Males)	0.93 ± 0.02	0.93 ± 0.03	0.856
Waist /Hip ratio (Females)	0.93 ± 0.03	0.92 ± 0.04	0.644

Pulmonary functions like FVC, FEV_1, FEV_1% and PEF were compared between cases and controls in males (Table 2) and females (Table 3). There was statistically significant decrease in the FVC, FEV_1 and PEF values in cases in both males and females.

Table-2: Comparison of pulmonary functions between cases & controls in males

Pulmonary function	Cases	Controls	P value
FVC (% predicted)	88.28 ± 14.93	102.28 ± 6.57	0.001*
FEV_1 (% predicted)	86.11 ± 16.75	95.78 ± 6.63	0.029*
FEV_1 % (% predicted)	96.22 ± 8.81	92.94 ± 2.75	0.886
PEF (% predicted)	75.33 ± 28.78	94.00 ± 9.68	0.013*
*statistically significant			

Table-3: Comparison of pulmonary functions between cases & controls in females

Pulmonary function	Cases	Controls	P value
FVC (% predicted)	87.33 ± 16.90	116.06 ± 10.83	0.001*
FEV_1 (% predicted)	79.93 ± 15.77	104.07 ± 8.01	0.001*
FEV_1 % (% predicted)	91.93 ± 14.06	89.53 ± 7.88	0.576
PEF (% predicted)	69.27 ± 20.80	97.53 ± 13.59	0.001*
*statistically significant			

Discussion

In our study, there was a significant decrease in the FVC, FEV_1 and PEF values in male and female GERD patients without respiratory manifestations. These results are suggestive of restrictive and obstructive airway changes. The obstructive changes can be explained on the basis of changes due to microaspiration of contents from GIT and vagally mediated bronchospasm. The aspiration of gastric content into respiratory passage triggers inflammation with a resultant narrowing of the passage. Chronic reflux may cause the inflammation of esophagus resulting in cough and bronchospasm mediated through neurogenic mechanism [15]. It is evident from the earlier studies that GERD, chronic cough or asthma co-exist. Different groups have tried to establish the cause, consequence relationship between these two conditions. Sontag and Havemann have reported that GERD symptoms are more common in asthmatics when compared to non-asthmatics suggesting that asthma can cause GERD [2, 8]. Asthmatics had more frequent day and night time reflux symptoms and greater incidence of reflux related nocturnal sleep disturbances. The protective responses become less active during night resulting in slower gastric emptying, decreased salivary production, decreased swallowing frequency and voluntary clearing behavior. This prolongs presence of food in the stomach and enhances the chances of aspiration of gastric contents into upper airway. During deep sleep, the esophageal sphincter pressure decreases and fails to prevent the gastric reflux from esophagus to upper airway. The subjects, who have developed respiratory manifestations like asthma, develop higher negative intrapleural pressure and hence trans-diaphragmatic pressure gradient facilitating reflux. Thoracic distension and air trapping results in impairment of diaphragmatic functions. The impairment of diaphragmatic function can produce the restrictive airway changes in asymptomatic subjects with GERD. Sontag et al have reported prevalence of esophagitis in 40 % of asthmatic subjects [5]. Wu et al have demonstrated airway hyper-responsiveness with esophageal acid perfusion without a significant change in the pulmonary functions in asthmatics [16].

It is evident from our study that GERD patients without respiratory symptoms also have altered pulmonary function. It is reasonable to suggest GERD produces changes in respiratory function and can aggravate the respiratory symptoms in asthmatics. This relationship has to be considered and a prompt treatment has to be instituted to prevent development of asthma like symptoms in GERD. Further, during the treatment of bronchial asthma, efforts have to be made to explore the presence of GERD. Treatment of co-existing GERD can go a long way is the effective management of bronchial asthma. A further study with larger sample size, correlating the duration of disease and extent of alteration in lung functions will help better understanding and effective management of GERD and associated respiratory problems.

References

1. Vakil N, Van Zanten SV, Kahrilas P, Dent J, Jones R. The Montreal definition and classification of gastroesophageal reflux disease: a global evidence based consensus. *Am J Gastroenterol* 2006; 101: 1900-120.

2. Havemann BD, Henderson CA, El-Serag HB. The association between gastroesophageal reflux disease and asthma: a systematic review. *Gut* 2007; 56: 1654-64 9.
3. Mathew JL, Singh M, Mittal SK. Gastro-esophageal reflux and bronchial asthma: current status and future direction. *Postgrad Med J* 2004; 80: 701-5.
4. Field SK. A critical review of studies of the effects of simulated or real gastroesophageal reflux on pulmonary function in asthmatic adults. *Chest* 1999; 115: 848-56.
5. Sontag SJ, Schnell TG, Miller TQ et al. Prevalence of esophagitis in asthmatics. *Gut* 1992; 33: 872-6.
6. Kiljander TO, Laitinen JO. The prevalence of gastroesophageal reflux disease in adult asthmatics. *Chest* 2004; 126: 1490-4.
7. Harding SM, Guzzo MR, Richter JE. The prevalence of gastroesophageal reflux in asthma patients without reflux symptoms. *Am J Respir Crit Care Med.* 2000; 162:34-9 7.
8. Sontag SJ, O'Connell, Miller TQ, Bernsen M, Seidel J. Asthmatics have more nocturnal gasping and reflux symptoms than nonasthmatics, and they are related to bedtime eating. *Am J Gastroenterol* 2004; 99: 789-96.
9. Ford AC, Forman D, Moayyedi P, Morice AH. Cough in the community; a cross sectional survey and relationship to gastrointestinal symptoms. *Thorax* 2006; 61: 975-9
10. Nordenstedt H, Nilsson M, Johansson S et al. The relation between gastroesophageal reflux and respiratory symptoms in population based study; the Nord-Trondelag health survey. *Chest* 2006; 129: 1051-6.
11. Gialson T, Janson C, Vermeire P. Respiratory symptoms and nocturnal gastroesophageal reflux. A population-based study of young adults in three European contries. *Chest* 2002; 121: 158-63.
12. ICMR. Ethical guidelines for Biomedical research on human participants. 2006.
13. Lund ell L, Dent J, Bennett j, et al. Endoscopic assessment of esophagitis: Clinical and functional correlates and further validation of Los Angeles classification. *Gut* 1999; 45: 172-80.
14. American Thoracic Society. Standardization of spirometry. 1994 update. *Am J Respir Crit Care Med* 1995; 152: 1107-36.
15. Irwin RS, Richter JE. Gastroesophageal reflux and chronic cough. *Am J. Gastroenterol* 2000; 95: S9-14.
16. Wu D-N, Tanifuji Y, Kobayashi H. Effects of esophageal acid perfusion on airway hyperresponsiveness in patients with bronchial asthma. *Chest* 2000; 118: 1553-6.

*All Correspondence: Dr. D. Venkatesh, Professor of Physiology, M.S. Ramaiah Medical College, Bangalore–560 054 Karnataka, India Email: venkatesh40@gmail.com