

Analysis of Prevalence Rate of Gastroesophageal Reflux Symptoms in Patients with Both Acute and Nonacute Cough

Himanshu Dutt Sharma¹, Sarvesh Kumar^{2*}

¹Assistant Professor, Department of General Medicine, Saraswathi Institute of Medical Sciences, Hapur Road, Anwarpur, Uttar Pradesh, India. ²Assistant Professor, Department of General Medicine, Krishna Mohan Medical College and Hospital, Mathura, Uttar Pradesh, India.

ABSTRACT

Introduction: Gastroesophageal reflux disease or GERD is a digestive disorder that most commonly associated with the ring of muscles present between oesophagus and stomach. This ring of muscle is commonly called as the lower oesophageal sphincter (LES). If an individual developed this kind disorders, he/she might experience heartburn or acid ingestion. Gastrooesophageal reflux disease (GERD) symptoms are more common in asthmatic individual. There are some evidence reporting that the severity and frequency of GERD symptoms are directly related to the severity of asthmatic condition.

Materials and Methods: This study was set to be carried out in 1500 consecutive patients (788 men and 937 women with a mean age of 42.2 years) who were reported to the outpatient ward in the Department of General Medicine, Saraswathi Institute of Medical Sciences, Hapur Road, Anwarpur, Uttar Pradesh (India) were taken for the present study. A written informed consent was taken from all of the patients. Inclusion criteria formulated were no patients had previous history of the administration of proton pump inhibitor (PPI), H2-receptor antagonist, antibiotics, steroids or nonsteroidal antiinflammatory drugs for a period of at least two months before the beginning of investigation. When the cough lasts for more than 3 weeks, it is considered as non-acute cough in the present study.

Results: On the whole, 656 (38%) patients were diagnosed as GERD and 230 (13%) had respiratory symptoms including cough, sputum, and/ or dyspnoea. GERD symptoms were found in 104 of 230 patients with respiratory symptoms and in 554 of 1500 patients without respiratory symptoms. There were no significant differences in age, male to female ratio, or the proportion of hypertension treated and current smokers between patients with and without respiratory symptoms.

INTRODUCTION

Gastroesophageal reflux disease or GERD is a digestive disorder that most commonly associated with the ring of muscles present between oesophagus and stomach. This ring of muscle is commonly called as the lower oesophageal sphincter (LES). If an individual developed this kind disorders, he/she might experience Among 230 patients with respiratory symptoms, 117 were diagnosed as acute cough and had a F-scale score of 6.9 ± 6.4 . The remaining 113 patients were considered as having non-acute cough and had a F-scale score of 7.8 ± 6.3 . There was no observable significant difference in F-scale score between the two groups. 43 (37%) of 117 patients with acute cough and 48 (43%) of 113 with nonacute cough had GERD symptoms. The difference in prevalence of GERD between acute and nonacute cough groups did not attain statistical significance possibly.

Conclusion: To conclude, patients with respiratory symptoms are at a significantly increased risk of developing GERD. There was no observable difference in GERD prevalence between the patients with acute and nonacute cough which suggests that the development of GERD is commonly associated with the presence of respiratory diseases without the development of respiratory symptoms.

Keywords: GERD, Asthmatic, Acute, Chronic Cough.

*Correspondence to: Dr. Sarvesh Kumar, Assistant Professor, Department of General Medicine, Krishna Mohan Medical College and Hospital, Mathura, Uttar Pradesh, India.

Article History:

Received: 19-12-2017, Revised: 11-01-2018, Accepted: 22-01-2018

Access this article online				
Website: www.ijmrp.com	Quick Response code			
DOI: 10.21276/ijmrp.2018.4.1.153				

heartburn or acid ingestion. Gastro-oesophageal reflux disease (GERD) symptoms are more common in asthmatic individual. Systematic review on GERD symptoms evaluation in asthmatics has reported a prevalence rate ranging from 30-90% when compared the same with general population which ranges

between 10-20%.1 A study conducted from South East Asia observed the prevalence of GERD symptom of about 57% in asthmatics when compared to 34% in non-asthmatic individuals,² and a urban based study on Indian population reported that 74.8% of asthmatics had a history of GERD during their disease course.³ There are some evidences reporting that the severity and frequency of GERD symptoms are directly related to the severity of asthmatic condition.⁴⁻⁶ The drugs which should be given to asthmatics might predispose this condition or if present already it may worsen the GERD symptoms.7-11 In addition to the higher prevalence of GORD symptoms, asthmatics might also associate their GERD symptoms with respiratory symptoms and the socalled condition is referred as Reflux Associated Respiratory Symptoms (RARS). Various studies report the chronic respiratory manifestations of GERD.¹² While others reported a relation between reflux and cough and reflux & bronchial hyperresponsiveness.13-16

Various studies showed the association between the GERD and asthmatics had selection bias since data recorded were in a retrospective manner. Whereas certain studies which addresses the association between respiratory disorders and GERD symptoms in a randomly selected group of persons are sparse. The incidence of chronic bronchitis, asthma, and chronic pulmonary disease is 1.5 times greater in patients with reflux esophagitis.^{17,18} While various other studies elucidated a high prevalence of GERD among asthmatic patients.^{19,20} Hence, even-

after the availability of various studies that encased a close relation between GERD symptoms and chronic cough, it has still been unknown to identify whether acute cough is also associated with GERD. The aim of this study was to assess the relationship between GERD and respiratory symptoms in general practice.

MATERIALS AND METHODS

This study was set to be carried out in 1500 consecutive patients (788 men and 937 women with a mean age of 42.2 years) who were reported to the outpatient ward in the Department of General Medicine, Saraswathi Institute of Medical Sciences, Hapur Road, Anwarpur, Uttar Pradesh (India) were taken for the present study. A written informed consent was taken from all of the patients. Inclusion criteria formulated were no patients had previous history of the administration of proton pump inhibitor (PPI), H2-receptor antagonist, antibiotics, steroids or nonsteroidal anti-inflammatory drugs for a period of at least two months before the beginning of investigation. Patients who had a previous history of partial gastrectomy were excluded from the study. The study was carried out in accordance with the Declaration of Helsinki and was priorly approved by the institutional ethical committee. When the cough lasts for more than 3 weeks, it is considered as non-acute cough in the present study. All values are denoted as mean ± SD. Comparisons of groups were made using Student's t test or chisquare tests whichever is appropriate. P - value of less than 0.05 was considered as statistically significant.

Table 1: Comparison of GERD prevalence and F-scale score between patients

	1		
Parameters	Respiratory symptoms (+)	Respiratory symptoms (-)	P - value
No. of patients	230	1500	
Age (years)	42.2 ±9.3	43.4 ±8.7	-
Male to female ratio	98:134	694:807	-
Current smoker	80 (34.7%)	488 (32.5%)	-
Hypertension	18 (7.8%)	114 (7.6%)	-
F – scale score	7.5 ±6.4	4.6 ±5.9	<0.01
No. of GERD	106 (46%)	554 (36.9%)	<0.01

with and without respiratory symptoms

Table 2: Comparison of GERD prevalence and F-scale score between patients

	with and acute and nonacute cough			
Parameters	Acute cough	Non-acute cough	P – value	
No. of patient	117	113		
F – scale score	6.9±6.4	7.8±6.3	-	
No. of GERD	45 (38.4%)	50 (44.2%)		

Table 3: The GERD prevalence in patients with and without pharyngeal symptoms in

acute and nonacute cough groups

Parameters	Non-acute cough		Acute cough				
Pharyngeal symptoms	+	-	+	-			
No. of patient	26	87	55	59			
No. of GERD	16 (61.5%)	33 (37.9%)	23 (41.8%)	20 (33.8%)			

RESULTS

On the whole, 656 (38%) patients were diagnosed as GERD and 230 (13%) had respiratory symptoms including cough, sputum, and/ or dyspnoea. GERD symptoms were found in 104 of 230 patients with respiratory symptoms and in 554 of 1500 patients without respiratory symptoms. Patients who were reported with respiratory symptoms had developed GERD symptoms more frequently than patients without respiratory symptoms with a statistical significance (p < 0.01, Table 1). F-scale score is significantly higher in patients with respiratory symptoms (7.5 \pm 6.4) than in those without respiratory symptoms (4.6 \pm 5.9) (p < 0.01). There were no significant differences in age, male to female ratio, or the proportion of hypertension treated and current smokers between patients with and without respiratory symptoms (Table 1). Among 230 patients with respiratory symptoms, 117 were diagnosed as acute cough and had a F-scale score of 6.9±6.4. The remaining 113 patients were considered as having non-acute cough and had a F-scale score of 7.8±6.3. There was no observable significant difference in F-scale score between the two groups. 43 (37%) of 117 patients with acute cough and 48 (43%) of 113 with nonacute cough had GERD symptoms (Table 2). The difference in prevalence of GERD between acute and nonacute cough groups did not attain statistical significance possibly. Comparison of the prevalence of GERD between the patients with and without pharyngeal symptoms was tabulated in Table 3. In patients with nonacute cough, those were found in 16 (61.5%) of 26 patients with pharyngeal symptoms and in 33 (38%) of 87 without them. For acute cough, 23 (41.8%) of 55 patients with pharyngeal symptoms, and 20 (31%) of 59 patients without pharyngeal symptoms were diagnosed as GERD. The differences in the prevalence of GERD between patients with and without pharyngeal symptoms did not reach a statistical significance in acute and nonacute cough groups.

DISCUSSION

GERD is a common entity which refers to the abnormal exposure of the oesophageal mucosa to gastric contents especially the acids secreted. Various population based surveys have reported typical GERD signs and symptoms which include heartburn and acid regurgitation, at least yearly prevalence ranging from 26% to 60%.^{21,22,23} These results are in concordance with our findings where GERD symptoms in the general population are usually less common in the east when compared to the western region.24,25 Helicobacter pylori infection most probably result in the condition called hypochlorhydria which is more commonly seen in individuals with atrophic gastritis and Patients affected by H. pylori were proved to be at less risk of developing GERD,²⁶ which is the same generally observed in Japan with high prevalence of H. pylori infection. Moreover, several reports showed an increase in the trend of GERD in Asian countries over the recent years.24,25 Although it has been believed that the low acid secretion would be related mostly with a lower prevalence of GERD in the Far Eastern region. Kinoshita and colleagues (1997) briefed that gastric acid secretion in Japan is greatly rising in both H. pyloripositive and negative individuals. Actually, the decreasing prevalence of H. pylori infection was seen in many countries27,28,29 as well as in Japan.30

On the other hand, there have been many researches that have showed a close relation between GERD symptoms and chronic cough.^{18,19,20} And several possible mechanisms were also explained underlying a relation between GERD and respiratory symptoms. Two mechanisms that have been postulated through which GERD may induce cough. They are micro-aspiration of gastric refluxate into the lung causing an exudative mucosal secretion and secondly, a vagally mediated distal oesophagealtracheobronchial reflex which ultimately leads to bronchospasm.³¹ The foregut and the respiratory tract have more common embryological origins and share the same number of reflexes. The existence of a vagally mediated esophageal-tracheobronchial reflex could possibly explain the reason behind worsening of asthmatic condition after a large meal. ³² Micro-aspiration has also postulated as a cause of GERD induced bronchospasm. In guinea pig model, acid perfusion of the oesophagus has caused neurally mediated airway inflammation of the bronchus.33 The intimate mechanisms of acid-induced airway obstruction are majorly dependent on the activation of capsaicin-sensitive sensory nerves with the subsequent release of tachykinins which modulate wide area of airway dysfunction.33 The study carried out by Wilson and colleagues (1987) observed direct proof that ingestion of 200 mL of 0.1 N Hcl increased bronchial activity. Thus, the heightened bronchial reactivity, micro-aspiration and a vagally mediated reflex mechanism are the reported possible pathways in the earlier researches.

The increased respiratory effort and repeated cough augment the abdominal pressure which facilitates the retrograde movement of all the gastric contents.³⁴ Changes associated with the lung volume may change the relationship between the diaphragm and lower oesophageal sphincter (LES) that is the ring muscles disrupting the normal physiological activity.7 The effect of asthmatic medications as well as the negative intrathoracic pressure may be produced during the asthma attacks which may overcome the protective nature of the LES, resulting in increased GERD signs and symptoms.20 Theophylline which is a bronchodilator, has been effectively shown to induce the gastric acid secretion and lower the LES pressure.35 Unavoidable exposure to small amounts of acid has been documented to be resulted in the impaired laryngopharyngeal sensitivity and thereby potentially increasing the risk of aspiration.³⁵ Using prolonged oesophageal pH monitoring, persistent cough without obvious pulmonary aetiology was most likely to be associated with the episodes asymptomatic gastroesophageal reflux.34 Similarly, cough due to any reason can precipitate gastroesophageal reflux.31 Based on these, the present study was done to evaluate the relationship between GERD symptoms and acute cough was postulated. Similarly, to the results observed previous studies,18,19,20 patients with respiratory symptoms developed the symptoms of GERD more frequently than patients without respiratory symptoms which reported a statistical significance in the present study. Although the GERD prevalence is significantly higher in patients with non-acute cough than in those with acute cough since there was no significant difference. This is believed to be that even acute cough can cause an increase in intraabdominal pressure which indirectly might promote gastric contents movements into the oesophagus. It seems falsely that GERD might cause acute cough since GERD is a chronic disease. Cough induced by GERD should be prolonged, persistent and chronic.

Sometimes few patients complain about having persistent cough following the symptoms of an upper respiratory tract infection in the routine clinical practice. When cough has been reported to be present for at least 3 weeks, but not more than 8 weeks, it is considered as a diagnosis of postinfectious cough.³⁶

The frequency of postinfectious cough increases up to 25% to 50%³⁶ and gastroesophageal reflux is thought to be one of its possible mechanisms which suggests that medical treatment of acid reflux in acute phase of cough might reduce the postinfectious cough even though the pathogenesis is often multifactorial usually.

CONCLUSION

To conclude, patients with respiratory symptoms are at a significantly increased risk of developing GERD. There was no observable difference in GERD prevalence between the patients with acute and nonacute cough which suggests that the development of GERD is commonly associated with the presence of respiratory diseases without the development of respiratory symptoms. It is more important that the association between GERD and acute cough could be more vastly recognized by the primary care physicians.

REFERENCES

1. Havemann BD, Henderson CA, El-Serag HB: The association between gastro-oesophageal reflux disease and asthma: a systematic review. Gut 2007, 56:1654-64.

2. Chunlertrith K, Boonsawat W, Zaeoue U: Prevalence of gastrooesophageal reflux symptoms in asthma patients at Srinagarind Hospital. J Med Assoc Thai 2005, 88:668-71.

3. Gopal B, Singhal P, Gaur SN: Gastro-oesophageal reflux disease in bronchial asthma and the response to omeprazole. Asian Pac J Allergy Immunol 2005, 23:29-34.

4. Nakase H, Itani T, Mimura J, Kawasaki T, Komori H, Tomioka H, Chiba T: Relationship between asthma and gastrooesophageal reflux: significance of endoscopic grade of reflux oesophagitis in adult asthmatics. J Gastroenterol Hepatol 1999, 14:715-22.

5. Gatto G, Peri V, Cuttitta G, Cibella F: Gastro-oesophageal reflux: prevalence in asthmatics in Italy. Dig Liver Dis 2000, 32:75.

6. Shimizu Y, Dobashi K, Kobayashi S, Ohki I, Tokushima M, Kusano M, Kawamura O, Shimoyama Y, Utsugi M, Mori M: High prevalence of gastro-oesophageal reflux disease with minimal mucosal change in asthmatic patients. Tohoku J Exp Med 2006; 209:329-36.

7. Stein MR, Towner TG, Weber RW, Mansfield LE, Jacobson KW, McDonnell JT, Nelson HS: The effect of theophylline on the lower esophageal sphincter pressure. Ann Allergy 1980, 45:238-41.

8. Berquist WE, Rachelefsky GS, Kadden M, Siegel SC, Katz RM, Mickey MR, Ament ME: Effect of theophylline on gastrooesophageal reflux in normal adults. J Allergy Clin Immunol 1981, 67:407-11.

9. Harding SM, Richter JE: The role of gastro-oesophageal reflux in chronic cough and asthma. Chest 1997, 111:1389-402.

10. Crowell MD, Zayat EN, Lacy BE, Schettler-Duncan A, Liu MC: The effects of an inhaled beta (2)-adrenergic agonist on lower esophageal function: a dose-response study. Chest 2001, 120:1184-9.

11. Lazenby JP, Guzzo MR, Harding SM, Patterson PE, Johnson LF, Bradley LA: Oral corticosteroids increase esophageal acid contact times in patients with stable asthma. Chest 2002, 121:625-34.

12. Róka R, Rosztóczy A, Izbéki F, Taybani Z, Kiss I, Lonovics J, Wittmann T: Prevalence of respiratory symptoms and diseases associated with gastroesophageal reflux disease. Digestion 2005, 71:92-6, Epub 2005 Mar 16.

13. Foroutan HR, Ghafari M: Gastroesophageal reflux as cause of chronic respiratory symptoms. Indian J Pediatr 2002, 69:137-9.

14. Maher MM, Darwish AA: Study of respiratory disorders in endoscopically negative and positive gastroesophageal reflux disease. Saudi J Gastroenterol 2010, 16:84-9.

15. Matsumoto H, Niimi A, Takemura M, Ueda T, Yamaguchi M, Matsuoka H, Jinnai M, Chin K, Mishima M: Prevalence and clinical manifestations of gastro-oesophageal reflux-associated chronic cough in the Japanese population. Cough 2007, 3:1-4.

16. Takenaka R, Matsuno O, Kitajima K, Ono E, Hiroshige S, Nishitake T, Miyazaki E, Kumamoto T: The use of frequency scale for the symptoms of GORD in assessment of gastro-oesophageal reflex symptoms in asthma. Allergol Immunopathol (Madrid) 2010, 38(1):20-4, Epub 2010 Jan 25.

17. Kennedy TM, Jones RH, Hungin AP, O'Flanagan H, Kelly P: Irritable bowel syndrome, gastro-oesophageal reflux, and bronchial hyperresponsiveness in the general population. Gut 1998, 43(6):770-774.

18. el Serag HB, Sonnenberg A. 1997. Comorbid occurrence of laryngeal or pulmonary disease with esophagitis in United States military veterans. Gastroenterology, 113:55–760.

19. Sontag SJ, O'Connell S, Khandelwal S, et al. 1990. Most asthmatics have gastroesophageal reflux with or without bronchodilator therapy. Gastroenterology, 99:613–20.

20. Vaezi MF. 2003. Extraesophageal manifestations of gastroesophageal refl ux disease. Clin Cornerstone, 5:32–8.

21. Ruth M, Mansson I, Sandberg N. 1991. The prevalence of symptoms suggestive of esophageal disorders. Scand J Gastroenterol, 26:73–81.

22. Stanghellini V. 1999. Three-month prevalence rates of gastrointestinal symptoms and the influence of demographic factors: results from the Domestic/International Gastroenterology Surveillance Study (DIGEST). Scand J Gastroenterol, 231(Suppl):20–8.

23. Kennedy T, Jones R. 2000. The prevalence of gastroesophageal refl ux symptoms in a UK population and the consultation behavior of patients with these symptoms. Aliment Pharmacol Ther, 14:1589–94.

24. Ho KY, Chan YH, Kang JY. 2005. Increasing trend of reflux esophagitis and decreasing trend of Helicobacter pylori infection in patients from a multiethnic Asian country. Am J Gastroenterol, 100:1923–8.

25. Chen M, Xiong L, Chen H, et al. 2005. Prevalence, risk factors and impact of gastroesophageal reflux disease symptoms: A population-based study in South China. Scand J Gastroenterol, 40:759–67.

26. Mishima I, Adachi K, Arima N, et al. 2005. Prevalence of endoscopically negative and positive gastroesophageal refl ux disease in the Japanese. Scand J Gastroenterol, 40:1005–9.

27. Banatvala N, Mayo K, Megraud F, et al. 1993. The cohort effect and Helicobacter pylori. J Infect Dis, 168:219–21.

28. Kosunen TU, Aromaa A, Knekt P, et al. 1997. Helicobacter antibodies in 1973 and 1994 in the adult population of Vammala, Finland. Epidemiol Infect, 119:29–34.

29. Roosendaal R, Kuipers EJ, Buitenwerf J, et al. 1997. Helicobacter pylori and the birth cohort effect: evidence of a continuous decrease of infection rates in childhood. Am J Gastroenterol, 92:1480–2.

30. Asaka M, Kimura T, Kudo M, et al. 1992. Relationship of Helicobacter pylori to serum pepsinogens in an asymptomatic Japanese population. Gastroenterology, 102:760–6.

31. Ing AJ, Ngu MC, Breslin AB. 1994. Pathogenesis of chronic persistent cough associated with gastroesophageal refl ux. Am J Respir Crit Care Med, 149:160–7.

32. Pellicano R, Ponzetto A, Smedile A, et al. 2005. Gastroesophageal refl ux disease and asthma: would be possible to improve therapy on the basis of what is now known? Panminerva Med, 46:135–40.

33. Ricciardolo FL, Gaston B, Hunt J. 2004. Acid stress in the pathology of asthma. J Allergy Clin Immunol, 113:610–19.

34. Field SK, Field TS, Cowie RL. 2001. Extraesophageal manifestations of gastroesophageal refl ux. Minerva Gastroenterol Dietol, 47:137–50.

35. Harding SM. 2001. Gastroesophageal refl ux, asthma, and mechanisms of interaction. Am J Med, 111(Suppl):S8–S11.

36. Davis SF, Sutter RW, Strebel PM, et al. 1995. Concurrent outbreaks of pertussis and Mycoplasma pneumoniae infection: clinical and epidemiological characteristics of illnesses manifested by cough. Clin Infect Dis, 20:621–8.

Source of Support: Nil.

Conflict of Interest: None Declared.

Copyright: © the author(s) and publisher. IJMRP is an official publication of Ibn Sina Academy of Medieval Medicine & Sciences, registered in 2001 under Indian Trusts Act, 1882.

This is an open access article distributed under the terms of the Creative Commons Attribution Non-commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

Cite this article as: Himanshu Dutt Sharma, Sarvesh Kumar. Analysis of Prevalence Rate of Gastroesophageal Reflux Symptoms in Patients with Both Acute and Nonacute Cough. Int J Med Res Prof. 2018 Jan; 4(1): 684-88. DOI:10.21276/ijmrp.2018.4.1.153