

ASSESSMENT OF CORRELATION BETWEEN OBESITY AND BARRETT'S ESOPHAGUS IN A POPULATION OF URBAN AREAS OF UDAIPUR

Tambi Shyam Sharan¹, Khoiwal Rajesh^{2*}

1. Assistant professor ,Department of General Medicine, Geetanjali Medical College and Hospital ,Udaipur , Rajasthan, India
2. Assistant professor ,Department of General Medicine, Geetanjali Medical College and Hospital ,Udaipur , Rajasthan, India

*Email of corresponding author: shyamstambi@gmail.com

Received: 12/12/2013

Revised: 21/04/2014

Accepted:29/04/2014

Abstract

Background: - The main risk for Barrett's esophagus (BE) is considered to be gastro esophageal reflux disease (GERD). The correlation between obesity and Barrett's esophagus (BE) in urban areas of Udaipur {Rajasthan} is studied in present study in patient visiting Geetanjali Medical College, Udaipur.

Material & method:- A cross-sectional study had been carried out from January 2013 to September 2013 at Geetanjali Medical College & Hospital, Udaipur in which 1080 subjects who underwent upper gastrointestinal endoscopy and the prevalence of endoscopically suspected BE was evaluated. Obesity was evaluated by body mass index (BMI, $\geq 25 \text{ kg/m}^2$) and waist circumference (WC) (males, $\geq 85 \text{ cm}$; females, $\geq 90 \text{ cm}$). Since the endoscopic diagnosis of short endoscopically suspected BE ($<1 \text{ cm}$ in extent) is difficult therefore it was excluded from the study. **Result:-** In present study by generating data and by analyzing it was came out that obesity (BMI, WC) is not independently a risk factor for barrett's oesophagus and results are non significant (p value >0.05). In contrast; the reflex eosophagitis (RE) significantly found as a risk factor (p value= 0.00) **Conclusion:-** Reflex eosophagitis but not obesity may have an independent association with the risk of endoscopically suspected BE. Furthermore, obesity measures were not independent risks for RE.

Keywords:- Barrett's esophagus, Body mass index, obesity, Reflux esophagitis

INTRODUCTION

The main risk for Barrett's esophagus (BE) is considered to be gastro esophageal reflux disease (GERD). (1) In present world there is an increase in prevalence of barrett's esophagus per decade and the incidence rates are also increasing. (2) In contrast few studies have been done before for association between RE and BE have found a positive association. (3)

Previous studies on the correlation between obesity and RE or BE have been limited to retrospective studies. (4) Hence, it is controversial whether obesity is actually an independent risk factor for BE or not. gastroesophageal junction , the distal end of the esophageal palisade vessels versus the proximal margin of the gastric folds. (5) BE diagnosis

requires histological confirmation of the specialized intestinal metaplasia (SIM). Thus, cases without histological confirmation of SIM are defined as endoscopically suspected BE (ESBE) in the US, but are not regarded as BE. (6) In most of the BE cases identified the lesions were very short, i.e., < 1 cm in length. Such cases are classified as ultra-short BE, although the diagnosis of this disease entity is difficult and highly unreliable. (7) Some previous studies have included patients who were taking proton pump inhibitors (PPIs), which may induce the regression and normalization of BE. Thus, the associations between obesity and BE should be investigated separately in patients taking and those not taking PPIs. (8)

MATERIAL & METHOD

The present study is a hospital based cross-sectional study had been carried out from January 2013 to September 2013 at Geetanjali Medical College & Hospital, Udaipur in which 1080 subjects who underwent upper gastrointestinal endoscopy and the prevalence of endoscopically suspected BE (ESBE) was evaluated. Most patients were out door patients, and patients who were previously undergone upper gastrointestinal tract surgery and who were previously undergone endoscopy and those who were followed in the surveillance of BE were excluded from the present study. BMI was calculated as weight divided by the square of height (kg/m^2). "Obesity" was defined as $\text{BMI} > 25.0 \text{ kg}/\text{m}^2$ (9) , Along with Waist Circumference based on the criteria (> 85 cm for men and > 90 cm for women) was defined as an abnormal WC.

In this study at our hospital the transnasal endoscopy with an ultrathin endoscope was performed. A standardized questionnaire was used to obtain a history from each subject regarding GERD symptoms, smoking and alcohol habits prior to endoscopy. The history of Proton pump inhibitors (PPIs) was asked by self-report and a review of the medical prescriptions in our database. From all the patients prior to this study a written informed consent was obtained. In the present study ESBE was diagnosed as columnar-lined epithelium between the lower end of the palisade vessels of the lower esophagus and the squamo-columnar junction, but if the palisade vessels could not be visualized, then the upper end of the gastric folds was regarded as the GEJ. When the ESBE length was considered to be 1 cm or more, the length was measured in comparison to the length of the endoscope reciprocally if the length was <1.0 cm, the ESBE length was judged in comparison to the biopsy forceps. Data were prospectively collected and evaluated in MS excel and SPSS version 16.

RESULT

About 1080 individuals participated in the present study with prior consent of subject himself. We included only 20 to 80 years individuals for the ease of study.

Out of them 570 were males and 510 were females. In the selected subjects 194 subject were not taking proton pump inhibitors and the rest of 886 subjects taking proton pump inhibitors.

Table 1: Distribution according to gender and age of the subjects.

Age (years)	Male (%)	Female (%)	Total (%)
20-40	160(28.1)	151(29.6)	311(28.8)
40-60	210(36.8)	174(34.1)	384(35.5)
60-80	200(35.1)	185(36.3)	385(35.6)
Total	570(100)	510(100)	1080(100)

All subjects evaluated on the basis of risk factors i.e. reflex esophagitis, and obesity by the mode of BMI and WC. Results have shown no significant association of obesity and ESBE in present study means (p value >0.05). In contrast the results were significant in those who were presented with RE means (p value = 0.00).

Table 2: Impact of obesity and RE on The occurrence of Barrett's esophagus in subjects based on taking and those who were not taking PPIs.

Subjects with Risk factor developing ESBE	Total no. of subjects examined for ESBE (n=1080)		P Value
	Not on PPIs (n=194)	On PPIs (n=886)	
RE	42(21.6%)	121(13.7%)	0.00
BMI >25,kg/m ²	36(18.6%)	134(15.1%)	0.23
WC>90,cm	03(0.01%)	13(0.01%)	1.00

The present study is a preliminary study and provides a snapshot of association between Barrett's esophagus and obesity along with reflux esophagitis, more elaborate pragmatic studies needed to get a clear picture of the correlation in urban and rural areas of Udaipur district.

DISCUSSION

A previous study reported that high WHR but not BMI, was associated with a significant increase in the risk of LSBE but not short-segment BE in white men (10). The present study is the hospital based cross-sectional study using endoscopic data collection to evaluate the association between obesity and the risk of ESBE, especially short-segment ESBE were investigated separately in subjects taking and those not taking PPIs in order to exclude the possibility of a drug affecting the pathophysiology of BE. RE is incompletely understood in its natural history because very few well-designed prospective studies and endoscopic studies in general populations have been performed (11). In the current study RE was a significant risk factor for ESBE development. In the present study clearly stated that RE was an independent risk factor for ESBE, while obesity was not a risk factor for RE. A previous study by Corley et al. says that differences in body fat distribution, rather than simple obesity as measured by the BMI, may cause GERD or BE, with abdominal fat deposition leading to an increase in intra-abdominal pressure and GERD, In contrast to that WC may predict the risk of BE or esophageal adenocarcinoma better than BMI

(12). The difference in the association between obesity and RE or BE might be explained, at least in part, by ethnic differences in the obesity pattern, especially the pattern of visceral adipose tissue deposition (4). One of the possible mechanism for the development of ESBE ≥ 1 cm in subjects may be that visceral obesity as measured by WC is associated with severe esophageal acid exposure that cannot be properly inhibited by PPIs treatment alone. That's why; 24-hour esophageal pH monitoring will be needed to further evaluate the mechanism in such patients. The WC in females differs from that in males because there is a difference in the preferential accumulation of VAT between females and males. Hence, the risk factors for BE development may be different between females and males. But, there was no association between obesity and RE and ESBE was found out in either females or males in the present study. Further elaborate studies and investigation required using a larger series of samples to determine the association between gender and BE. The present study had several potential limitations. A possible weakness is that only ESBE without histological confirmation of SIM was evaluated. Further more studies will be needed to accurately clarify the association. Second, present study was a hospital based study, so the pooled data might not represent the general population. In addition, with that, our sample size was relatively small. Third, it was not established whether any of the endoscopic BE patients had a hiatus hernia (may be a risk factor of BE development itself), H. pylori infection (may have an inverse association with BE). Along with that, we did not evaluate the administration of nonsteroidal anti-inflammatory

drugs, including aspirin, which have been significantly associated with a risk of BE (13).

CONCLUSION

In present study obesity were not risk factors for ESBE and it was found out that RE was the only one of the parameters studied that was a significant risk factor for ESBE independent of obesity. Further studies with a larger sample size will be needed to clarify the reason for this lack of association between obesity and ESBE in the urban and rural population of Udaipur district.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: The study was approved by the institutional ethics committee

REFERENCES

1. Sampliner RE: Updated guidelines for the diagnosis, surveillance, and therapy of Barrett's esophagus. *Am J Gastroenterol* 2002, 97:1888-1895.
2. El-Serag HG: Time trends of gastroesophageal reflux disease: a systematic review. *Clin Gastroenterol Hepatol* 2007, 5:17-26.
3. Anderson LA, The association between alcohol and reflux esophagitis, Barrett's esophagus, and esophageal adenocarcinoma, *Gastroenterology*.2009 Mar;136(3):799-805.
4. Visceral obesity and the risk of Barrett's esophagus in Japanese patients with non-alcoholic fatty liver disease. *BMC Gastroenterol* 2009, 9:56

5. The development and validation of an endoscopic grading system for Barrett's esophagus: the Prague C & M criteria. *Gastroenterology* 2006, 131:1392-1399.
6. Vakil N, van Zanten SV, Kahrilas P, Dent J, Jones R, Global Consensus Group: The Montreal definition and classification of gastroesophageal reflux disease: a global evidence-based consensus. *Am J Gastroenterol* 2006, 101:1900-1920.
7. Interobserver reliability in the endoscopic diagnosis and grading of Barrett's esophagus: an Asian multinational study. *Endoscopy* 2010 ,42:699-704.
8. Peters FT, Ganesh S, Kuipers EJ, Sluiter WJ, Klinkenberg-Knol EC, Lamers CB, Kleibeuker JH: Endoscopic regression of Barrett's oesophagus during omeprazole treatment; a randomised double blind study. *Gut* 1999, 45:489-494.
9. WHO BMI indices.(cited on 2013 dec22).available from who.int/bmi/index.jsp?introPage=intro_3.html
10. Kramer JR, Fischbach LA, Richardson P, Alsarraj A, Fitzgerald S, Shaib Y, Abraham NS, Velez M, Cole R, Anand B, Verstovsek G, Ruge M, Parente P, Graham DY, El-Serag HB: Waist-to-hip ratio, but not body mass index, is associated with an increased risk of Barrett's esophagus in white men. *Clin Gastroenterol Hepatol* 2013, 11:373-381.
11. Fullard M, Kang JY, Neild P, Poullis A, Maxwell JD: Systematic review: does gastro-oesophageal reflux disease progress. *Aliment Pharmacol Ther* 2006, 24:33-45
12. Corley DA, Kubo A, Levin TR, Block G, Habel L, Zhao W, Leighton P, Quesenberry C, Rumore GJ, Buffler PA: Abdominal obesity and body mass index as risk factors for Barrett's esophagus. *Gastroenterology* 2007, 133:34-41.
13. Omer ZB, Ananthakrishnan AN, Nattinger KJ, Cole EB, Lin JJ, Kong CY, Hur C: Aspirin protects against Barrett's esophagus in a multivariate logistic regression analysis. *Clin Gastroenterol Hepatol* 2012, 10:722-727