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Association Between Obesity and Asthma among Adults: A Hospital Based Case-Control Study

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Abstract: The aim is to study the association between obesity and asthma among adults by gender. The prevalences of both asthma and obesity have increased substantially in recent decades, leading to speculation that obese individuals might be at risk of asthma. However, the evidence of a relationship between obesity and asthma is not fully conclusive among adults. Hence we investigate the association between obesity and asthma among men and women using both measured weight and height and self-reported weight and height while controlling for the effects of the demographic and environmental factors. This case-control study involves a total of 159 adults; 53 cases and 106 controls enrolled in the month of July 2009 at PSG Hospitals. Body Mass Index (BMI) was calculated based on measured weight and height. The Odds Ratio (OR) with 95% confidence interval for obese individuals was estimated using logistic regression analysis with SPSS 11.5 for windows software (SPSS Inc., Chicago, Illinois). Multivariate logistic regression model was used to adjust all risk estimates for covariates. Obese women were found to have 9.14 times the risk of asthma than non obese women (95% confidence interval (CI):1.38, 35.68) after adjusting for covariates, such as age, education, environmental tobacco smoke and pet keeping. No significant association was observed among men although the direction of association is positive; adjusted odds ratio was 1.06 (95% CI: 0.12, 9.70). No significant association was observed between self-reported prevalence of obesity and asthma; among women, adjusted odds ratio = 4.33 (95% CI: 0.69, 27.37); and among men, adjusted odds ratio = 0.89 (95% CI: 0.11, 7.12). The study indicates a strong positive association between obesity and asthma among adult Indian women. The causal links between obesity and asthma by gender need to be further examined using prospective cohort studies.

Key words: Obesity, asthma, adults, males, females

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INTRODUCTION

The growing prevalence of obesity has been accompanied by rapidly rising asthma rates worldwide in both adults and children (Camargo *et al*., 1999; Ford, 2005; Nystad *et al*., 2004). A rapid increase in asthma in recent years cannot be ascribed to changes in genetic factors alone. The focus should be on behavioral and environmental factors. The parallel increase in the prevalence of obesity and asthma in the past three decades has led some researchers to postulate a causal relationship between the two conditions (Mathew *et al*., 2008; Shaheen, 1999; Sekar *et al*., 2005; Thomas *et al*., 2007). A number of studies have found a positive association between obesity and asthma in adults (Celedon *et al*., 2001; Chen *et al*., 1999) and in children (Von Mutius *et al*., 2001). Obesity has also been linked with impaired pulmonary function and airway hyper responsiveness (Gibson, 2000; Litonjua *et al*., 2002), but not in all studies (Sin *et al*., 2002). The most convincing evidence of a causal link between obesity and asthma in adults is provided by a large prospective cohort study of 85,911 nurses followed between 1991 and 1995, in which obese women were found to have a much greater risk of asthma and weight gain was positively associated with the risk of developing asthma (Camargo *et al*., 1999). Another recent prospective study of 10,597 adult twins in Finland followed for 9 years has found obesity to be associated with the risk of adult-onset asthma (Huovinen *et al*., 2003).

Convincing evidence of obesity causing asthma onset is also provided by the two prospective cohort studies of children. The Growing Up Today study of 16,862 children found that Body Mass Index (BMI) has a positive and independent risk of incident asthma in both boys and girls, concluding that increasing prevalence of obesity may partly explain the rising asthma prevalence in children (Camargo *et al*., 2003). The Children's Health Study in Southern California also found that overweight and obesity were associated with new-onset asthma in both boys and girls (Gilliland *et al*., 2003). Moreover, several prospective studies of obese asthmatics have shown that weight reduction leads to improved pulmonary function and reduced severity and frequency of asthma symptoms (Hakala *et al*., 2000; Stenius-Aarniala *et al*., 2000).

However, the evidence of a relationship between obesity and asthma is not fully conclusive among adults. Several studies found the relationship between obesity and asthma only in females, but not in males (Chen *et al*., 2002; Del-Rio-Navarro *et al*., 2003). Some found no relationship (Brenner *et al*., 2001) or fail to link increase in obesity to increase in asthma (Chinn and Rona, 2001). Some have also suggested a reverse relationship between asthma and obesity (Epstein *et al*., 2000).

Several studies have shown high correlation between self reported and measured BMI (Bolton-Smith *et al*., 2000; Niedhammer *et al*., 2000). The effect of misclassification of BMI in the association between gender was also reported in a recent study (Santillan and Camargo, 2003) and observed that the misclassification of BMI obscured the relationship between obesity and asthma to a greater extent among men than women, since obesity prevalence in the general population was higher among men. However, much of the research linking obesity and asthma to date has been carried out in developed countries. Most developing countries, with continuing high levels of undernutrition and high prevalence of communicable diseases, have paid little attention to the rapidly growing problems of obesity and asthma. This lack of attention is usually coupled with poor quality and availability of data on the two conditions. Only four studies in developing countries have associated overweight conditions with wheezing and asthma. One study in a high-altitude area in Korea linked high BMI to wheezing among the elderly (Jang *et al*., 2002). A second study in the

Anhui Province in China linked overweight condition with asthma in adults (Celedon *et al*., 2001) and a third study among Mexican adults linked measured obesity to asthma in both men and women (Santillan and Camargo, 2003). The fourth study by Mishra (2004) reported the effect of obesity among adult Indian women using data from India's Second National Family Health Survey (NFHS-2) conducted in 1998-99 and observed a strong positive association between obesity and asthma among adult Indian women.

These studies on the effects of obesity on asthma were based on the reported prevalence of asthma and not based on the physician's diagnosis and have not accounted the effect of environmental tobacco smoke at home and work, parental atopy and the effect of pet keeping which may confound the results. Similarly in many of these earlier studies, BMI was calculated based on the self reported weight and height and not based on the measured weight and height. Therefore, there is a further possibility of misclassification. Though asthma and obesity is on the rise in India, there is a paucity of data on the association between the two. Hence, we proposed to investigate the association between obesity and asthma among men and women using both measured weight and height and self-reported weight and height while controlling for the effects of the demographic and environmental factors.

MATERIALS AND METHODS

This case-control study involves a total of 159 adults, of which there were 53 adults enrolled from the outpatient clinic of Department of Tuberculosis and Chest Diseases of PSG Hospitals in the month of July 2009. The controls were 106 outpatients enrolled from the Department of Ear, Nose and Throat (ENT) and Department of Ophthalmology of PSG Hospitals who were free of the condition of interest. The Institutional Human Ethics Committee approved all subject recruitment and data collection procedures. A written informed consent was obtained from all the subjects.

Inclusion criteria for cases were as follows: (1) patients with documented diagnosis of persistent asthma (according to the Global Initiative for Asthma (GINA) guidelines) a period of at least 6 month prior to visit, (2) male and female patients aged greater than or equal to 20 years and (3) patients demonstrating an increase in FEV 1 of 12% or = 200 mLs within 30 min after administration of Short-Acting Beta Agonist (SABA). Alternatively, patient may have documentation of reversibility within the last 12 months. Exclusion criteria were as follows: (1) pregnant or nursing women, (2) patients who have smoked more than 10 pack years (pack year is calculated by multiplying the number of packs of cigarettes (1 pack has 20 cigarettes) smoked per day by the number of years the person has smoked) and (3) patients with a previous diagnosis of Chronic Obstructive Pulmonary Disease (COPD) (National Institutes of Health, Heart, Lung and Blood, 2002).

We measured the Socio Economic Status (SES) based on Prasad's modified classification based on Consumer Price Index (Industrial Work) (CPI (IW)) for the month of May 2009 after rounding off to the nearest Rs.10. For those with per capita income per month Rs.3330 and above were classified as Class 1 (Kumar, 1993). Physical activity levels were assessed based on the hours of physical exercise in a week (Bharathi *et al*., 2000). Height was measured in meters without shoes and weight was measured in kilograms with the subject wearing light clothing. BMI was computed for males and females using the formula: weight (kg)/height $(m)^2$. Subjects were classified as obese if their BMI was equal to or greater than 30 kg m^{-2} (Nystad *et al*., 2004). Their perceived weight and height were also collected. We also elicited whether they have any exposure to cigarette smoking by any member in the

household and in the work place and thereby exposure to environmental tobacco smoke was assessed. Parental atopy was defined as a history of maternal or paternal asthma, hay fever, allergic eczema and allergic conjunctivitis. Exposure to pets and its duration was assessed based on information on the presence of cats, dogs, birds, or other hairy animals during the past 12 months as well as more than 12 months (Jaakkola *et al*., 2002).

Statistical Analysis

The Odds Ratio (OR) with 95% confidence interval for obese individuals was estimated using logistic regression analysis with SPSS 11.5 for windows software (SPSS Inc., Chicago, Illinois). Multivariate logistic regression model was used to adjust all risk estimates for covariates. Possible covariates included were age, education, occupation, socioeconomic status, physical activity, environmental tobacco smoke at home, environmental tobacco smoke at work, eating patterns, parental atopy and pet keeping more than 12 months. The adjusted odds ratio and unadjusted odds ratios were calculated to evaluate the association of measured as well as self-reported obesity with asthma and analysis were stratified by gender. A p-value of <0.05 was considered statistically significant. The agreement between self reported obesity and measured obesity in men and women were estimated using kappa statistics.

RESULTS

Comparisons between cases and controls among males revealed no significant difference in terms of age, education, occupation, measured BMI, measured weight, prevalence of obesity based on measured as well as self reported BMI, socio economic status, eating patterns, duration of physical exercise, environmental tobacco smoke exposure. However, measured height, self-reported weight and self-reported height were significantly higher among controls. Among females, percentage of those having higher education was more in controls than in cases (Table 1). Other demographic and clinical characteristics

Table 1: Demographic and clinical characteristics of adults with and without asthma

	Males			Females		
Variables	Cases	Controls	p-value	Cases	Controls	p-value
Age	46.78±18.85	42.66±13.50	0.31	46.69±11.15	42.87±13.79	0.18
Education (college and above)	33.3	45.8	0.35	8.6	27.7	0.04
Occupation (professionals, land lord, home maker)	16.7	18.6	0.85	74.3	57.4	0.12
Measured body mass index (kg m^{-2})	23.65±4.47	23.79±4.01	0.89	25.57±4.71	24.30±4.32	0.21
Measured weight (kg)	62.88±10.79	67.27±12.15	0.17	60.43±12.38	57.29±10.60	0.22
Measured height (m)	1.63±0.05	1.68±0.06	0.00	1.53±0.06	1.54±0.06	0.93
Prevalence of obesity based on measured BMI	11.1	8.5	0.73	22.9	6.4	0.03
Self-reported weight (kg)	62.17±11.54	68.98±11.79	0.03	59.71±12.61	55.77±8.99	0.10
Self-reported height (m)	1.61±0.06	1.67±0.06	0.00	1.54±0.09	1.54±0.07	0.93
Prevalence of obesity based on self-reported measure BMI	11.1	11.9	0.93	20	6.4	0.08
Socio economic status (Class I)	94.4	93.2	0.85	82.9	76.6	0.49
Non vegetarian	94.4	94.9	0.94	88.6	85.1	0.65
Hours of physical exercise in a week	1.89±3.78	2.81±5.15	0.49	1.69±5.37	0.33±1.28	0.09
Environmental tobacco smoke at work place	44.4	47.5	0.82	22.9	25.5	0.78
Environmental tobacco smoke at home	33.3	22	0.33	31.4	29.8	0.87

Some data expressed as Mean±SD and other as percentage

revealed no significant difference between cases and controls. Briefly, of all the participants, 51.57% were females and 48.43% were males. The average age in years of men was 46.78 (standard deviation (SD), 18.75) and 42.66 (SD, 13.50) in cases and controls, respectively; among females it was 46.69 (SD, 11.15) and 42.87 (SD, 13.79), respectively. The average measured BMI (kg m^{-2}) among men in cases was 23.65 (SD, 4.47) and 24.20 (SD, 4.32) in controls; among females it was 25.57 (SD, 4.71) and 24.30 (SD, 4.32) respectively. The prevalence of obesity in men based on measured BMI was 11.1 and 8.5% in cases and controls, respectively; among females it was 22.9 and 6.4%, respectively and the prevalence of obesity in men based on self-reported weight and height was 11.1 and 11.9% in cases and controls, respectively; among females it was 20 and 6.4%, respectively.

Table 2 shows the effects of demographic and environmental factors on asthma. Higher education level was significantly associated to reduce the prevalence of asthma among females, odds ratio (OR) = 0.25 (95% CI: 0.06, 0.9) but not among males; OR = 0.59 (95% CI: 0.19, 1.79).

Table 3 shows the association between measured and self-reported obesity on asthma among men and women. Measured obesity was associated a significantly higher risk of asthma among women but not in men. The complete model adjusted for age, education, occupation, socio economic status, level of physical activity, environmental tobacco smoke at work, environmental tobacco smoke at home, eating habits, parental atopy and pet keeping shows females, who have obesity, had 9.14 times the risk of asthma than those non obese women (95% CI: 1.38, 35.68). However among men, a non significant association was observed; odds ratio is 1.06 (95% CI: 0.12, 9.70).

Table 2: Association of various risk factors on asthma by gender

	Male		Female	
Variable	OR	95% CI	OR	95% CI
Age	p = 0.35		p = 0.13	
20-29[a]	1		1	
30-39	0.41	0.08-2.79	0.95	0.18-5.08
40-49	1.22	0.24-6.32	3.11	0.67-14.44
50-59	0.46	0.07-3.02	5.33	0.89-31.92
60+	1.96	0.39-9.93	1.48	0.27-8.27
Education	p = 0.35		p = 0.04	
Up to secondary[a]	1		1	
College and above	0.59	0.19-1.79	0.25	0.06-0.9
Occupation	p = 0.62		p = 0.31	
Skilled labors[a]	1		1	
Clerk, teacher	0.39 (p = 0.19)	0.09-1.58	0.67 (p = 0.66)	0.11-4.19
Professionals, land lord	0.74 (p = 0.68)	0.17-3.19	0.67 (p = 0.74)	0.06-7.63
Home maker			2.08 (P = 0.18)	0.72-6.05
Socio economic status	p = 0.85		p = 0.49	
Class II, III, IV[a]	1		1	
Class I	1.24 (p = 0.85)	0.13-11.82	1.48 (p = 0.49)	0.48-4.48
Higher physical activity	0.96 (P = 0.49)	0.84-1.09	1.16 (p = 0.17)	0.93-1.45
Eating habits	p = 0.94		p = 0.65	
Vegetarian[a]	1		1	
Non vegetarian	0.91	0.08-9.33	1.36	0.36-5.05
Environmental tobacco smoke exposure at work	p = 0.82		p = 0.78	
No[a]	1		1	
Yes	0.89	0.31-2.56	0.86	0.31-2.4
Environmental tobacco smoke exposure at home	p = 0.33		p = 0.87	
No[a]	1		1	
Yes	1.77	0.56-5.63	1.08	0.42-2.71

[a]Reference category

Table 3: Effects of obesity on asthma among men and women

	Measured obesity				Self-reported obesity			
	Males		Females		Males		Females	
Model	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Model I: Unadjusted	1.35 (p = 0.73)	0.24, 7.63	4.35 (p = 0.04)	1.06, 17.81	0.93 (p = 0.93)	0.18, 4.92	3.67 (p = 0.07)	0.89, 15.37
Model II: Adjusted for age	1.24 (p = 0.80)	0.22, 9.64	4.68 (p = 0.03)	1.12, 19.52	0.92 (p = 0.91)	0.17, 4.89	3.66 (p = 0.07)	0.87, 15.49
Model III: Adjusted for age, education, occupation	1.54 (p = 0.64)	0.24, 9.64	5.71 (p = 0.03)	1.19, 27.51	1.34 (p = 0.74)	0.23, 7.83	4.25 (p = 0.07)	0.88, 20.64
Model IV[b] (Full model):	1.06 (p = 0.96)	0.12, 9.70	9.14 (p = 0.02)	1.38, 35.68	0.89 (p = 0.91)	0.11, 7.12	4.33 (p = 0.11)	0.69, 27.37

[b]Adjusted for age, education, occupation, socio economic status, physical activity, eating habit, exposure to pets, environmental tobacco smoke at work and home, and parental atopy

Since investigations of the association between obesity and asthma used both self-reported and measured weight and height, we conducted analyses between self-reported and measured weight and height. We also found a strong association between reported obesity and measured obesity among men and women. Among males, the reported prevalence of obesity was 11.7% and in females it was 12.2%, while the measured prevalence of obesity among males was 9.1% and in females 13.4%. The Kappa value among women was 61.8 and among men 72.2, which indicating a good level of agreement between self-reported and measured obesity. However, comparing the odds ratios, we found that the self-reported obesity underestimates the associations between obesity and asthma among men and women.

DISCUSSION

The study supports the hypothesis that obese women in India have substantially higher risk of asthma independent of their occupational status, level of physical activity, family history of asthma, environmental exposure to tobacco smoke at work place or inside the house, socioeconomic status, education levels, age, parental atopy and exposure to pet keeping inside and outside the house. These findings are consistent with growing evidence, from prospective cohort studies and provide further evidence from developing country settings that obesity may increase the risk of asthma in adult women (Camargo *et al*., 1999). The diagnosis of asthma was based on the diagnosis of physician which was clearly an added strength of our study compared to the self reported information in the previous studies (Mishra, 2004).

The interrelation between obesity and asthma may be bidirectional. Asthma may increase the risk of obesity due to a sedentary life style and obesity may increase the risk of asthma. The question deserves to be asked whether and how a person's asthma might be affected by excess weight. Some evidence suggests that obesity indeed affects the respiratory health status of patients with asthma (Wheeldon *et al*., 1994). The role of progesterone in the etiology of asthma also has been reported. In an earlier study, it was found that progesterone up-regulates beta-2 receptors. Also, the luteal phase increase in progesterone and estadiol is associated with an increased density of beta-2 adrenoreceptors on lymphocytes (Wheeldon *et al*., 1994). Similarly in another study, it was found that, a total of 40 mg of exogenously administrated progesterone caused an eightfold increase in the bronchorelaxant effect of the catecholamine isoprenaline (Foster *et al*., 1983). During the menstrual cycle, a time when asthma symptoms may worsen, Juniper *et al*. (1987) found no

change in nonspecific airway responsiveness. This suggests that airway inflammation is not changed but rather that adrenoreceptor regulation may be involved in asthma. In women, obesity influences hypothalamic-pituitary-ovarian function with increased androgen availability for peripheral aromatization to estrone. Obesity may also be associated with reduced levels of progesterone production (Deslypre, 1995). Leptin, a hormone related to the obesity gene, inhibits insulin-induced progesterone production (Spicer and Francisco, 1997). These facts lead one to speculate on a biologically plausible explanation for the association between obesity and asthma in women. Obesity reduces the levels of progesterone, causing down regulation of adrenoreceptors, thereby aggravating asthma control. In the US Nurses' Health Study, however, Troisi *et al.* (1995) found that the incidence of asthma was higher in postmenopausal women taking estrogens. In a longitudinal analysis based on the 89,061 women aged 27-44 years from the Nurses' Health Study, Camargo *et al.* (1999) found that the incidence of asthma was increased with increased baseline value of BMI during the 1991-1995 study period, which adds weight to the hypothesis that obesity causes asthma.

Similarly asthma can also cause obesity. Exercise and cold induced aggravation of asthma commonly occurs and may reduce the duration and intensity of physical activity, which is also a risk factor for obesity. None of the postulated rationales for an association between asthma and obesity can easily account for the observed gender difference in the obesity-asthma relationship, except the theory that obesity influences female sex hormones, which in turn, influences asthma control. Apart from the biochemical evidence, this theory is also supported by three clinical observations. Pregnancy and menstruation influence asthma control (McDonald and Burdon, 1996). It is also well documented that obesity influences sex hormones. Finally exogenous progesterone administration may play a role in asthma control in the premenstrual period (Dodge and Burrows, 1990).

There are some common risk factors for both asthma and obesity. Dietary risk factors for obesity are well known and have been postulated for asthma, including a relative excess of sodium and omega-6 fatty acids and a relative deficiency of antioxidant vitamins (Peat, 1996). A sedentary life-style may predispose to both obesity and increased indoor times, with a latter increased exposure to environmental tobacco smoke, pet and dust mite antigens, which are important risk factors for asthma.

Prospective studies conducted among adults are fairly consistent in finding that excess weight is associated with asthma. Of the eight prospective studies (Beckett *et al.*, 2001; Camargo *et al.*, 1999; Chen *et al.*, 1999; Ford *et al.*, 2004; Guerra *et al.*, 2002; Huovinen *et al.*, 2003; Shaheen *et al.*, 1999; Xu *et al.*, 2002), all but one have reported a positive association in either men, women, or both. Furthermore, weight gain is associated with an increased risk of asthma. The majority of large cross-sectional studies show that measures of excess weight (usually on the basis of BMI) are positively associated with the likelihood of having asthma, although in some studies the risk appears to be stronger among women than men (Chen *et al.*, 2002).

Present findings contradicts with the earlier findings of Santillan and Camargo (2003) that misclassification of BMI may lead to underestimate the association of asthma and obesity among men than women instead we observed that the self-reported measurements underestimate the associations in both men and women despite being found a fairly strong association between self-reported obesity and measured obesity among men and women.

Present study has several limitations. First, the period of the study was only one month and we could study only 53 cases and 106 controls and issues of sample size and power are important in generalizing our findings. Second, we studied only hospital outpatient asthma cases which do not represent those in the general population. Selection factors relating to

the study protocol could have created a fairly homogenous study group and for these reasons our findings may not be generalized to other socioeconomic strata and to other cultural and ethnic groups. Third, we could not measure the physical activity based on any validated physical activity questionnaire. Finally, in our study, obesity was calculated based on BMI. There are many other ways to measure excess weight. However, most studies in adults have used BMI, which is based on measurements of weight and height. Use of BMI to establish obesity has been endorsed by the World Health Organization (1998) and the National Institutes of Health (2002). Clearly, numerous other measures could also be used, such as waist circumference, waist/hip ratio, relative weight, skin folds, percentage of body fat calculated from dual-energy x-ray absorptiometry, bioelectrical impedance, underwater weighing and abdominal fat from computed tomographic scans. Though many of these measures show a high degree of correlation especially in increasingly obese societies (Deurenberg *et al*., 2002; Jackson *et al*., 2002), however it is unclear whether results from the existing studies on the basis of BMI would have produced findings that differed in important ways if other anthropometric measures other than BMI had been used (Snijder *et al*., 2006).

Choosing an appropriate set of confounding variables is not easy in a study on the association between obesity and asthma. To be a confounder, a variable has to be related to both the outcome and exposure variable and it is not clear what should constitute a minimal set of such variables. In our study, we have included age, sex, education, occupation, socio economic status, eating habit, physical activity, parental atopy, exposure to pets, environmental tobacco smoke at home and work place as potential confounders based on the previous literature (Ford, 2005).

Despite these limitations, the consistency in the size of crude and adjusted effects of obesity on asthma among women suggests a possible causal relationship. Moreover, in developing countries such as India, where data on BMI and clinical data on asthma are usually not available, data that we have collected provided a unique opportunity to study the relation between self reported and measured obesity with asthma. In addition, our study has several other strengths. First, the diagnosis of asthma was based on the standard guidelines by the physician and not based on the reported information. Second, information we have collected on parental atopy, exposure to pets, environmental tobacco smoke at work place and home and physical activity allowed us to examine the effects of obesity on asthma while controlling the effects of these variables. Finally, we have both measured and self-reported weight and height and investigated the effect of using both measures on the association between obesity and asthma.

CONCLUSION

In summary, we have found a significant positive association between obesity and asthma among females but not among males. The causal links between asthma and obesity by gender need to be further examined using prospective cohort studies as well as through large randomized clinical trials of weight loss, including bariatric studies, which may yield valuable insights into the obesity-asthma relationship, especially if rigorous definitions of asthma are used. Such research is especially important in the light of both the prevalence of obesity and asthma are rising rapidly in the developing countries. However, whether weight loss programs need to be tailored to patients with asthma deserves further consideration. Because the combination of energy restriction and increased energy output produces the most consistent long-term weight loss, good control of asthma is a necessity to allow many patients with this disorder to increase their level of physical activity. Health care

professionals can help to dispel any lingering doubts among most asthmatic patients about their ability to engage in adequate physical activity. Clearly, many aspects of obesity and asthma deserve further research.

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