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Journal search and commentary

Article reviewed: Obesity is a risk factor for asthma and wheeze but not airway hyperresponsiveness ☆

Mark H. Sanders

Division of Pulmonary, Allergy & Critical Care Medicine, University of Pittsburgh School of Medicine, Pittsburgh, PA, USA

Objective(s)

To determine if obesity is a risk factor for asthma, wheezing and shortness of breath, use of asthma medications or airway hyperresponsiveness (AHR) to an inhaled histamine challenge

Study design

Retrospective case review.

Study population

Data were pooled from the databases of three epidemiological studies that were conducted in New South Wales, Australia between 1991 and 1997. Information from 1971 randomly identified white adults was analyzed. No new data were collected for the purpose of the current report.

Methods

Through their participation in the parent epidemiological studies, individuals completed questionnaires, which among other items, provided information addressing 'recent wheeze' (defined as affirmation of wheezing during the preceding year), and 'recent asthma' (defined by the presence of recent wheezing plus having ever had physician diagnosed asthma).

Spirometric measures of pulmonary function including FEV_1 (the volume exhaled after 1 s in a forced expiratory maneuver), FVC (forced vital capacity defined by the maximal volume of air that is exhaled from total lung capacity) and the FEVIFVC ratio were also analyzed. Participants' response to a histamine inhalation challenge was also assessed. The histamine provocation test is one in which the individual is challenged with sequential inhalations of a histamine solution of progressively increasing concentrations with spirometric measurements after each concentration. The challenges are stopped, when the FEV_1 falls by 20% from baseline values. The histamine concentration at which this occurs is noted and defines the $PD_{20}FEV_1$. In addition, the investigators in this study calculated the dose response ratio (DRR), which is the percentage change from baseline FEV_1 divided by the histamine dose. For their analysis, the investigators defined a airway hyperresponsiveness (AHR) as a 20% reduction in FEV, at a histamine dose of $\leq$3.9 μmol (equivalent to a DRR: $>$8.1).

Body mass index (BMI) was calculated from the weight, kg/height, m^2. An overweight condition was defined as a BMI: 24.9–29.9; moderate obesity was reflected by a BMI: 30–34.9; severe obesity was reflected by a BMI: 35–39.9, and very severe obesity was defined by a BMI $\geq$40. In their analyses, the investigators combined the severe and very severe groups.

☆ Shachter LM, Salome CM, Peat JK, Woolcock AJ. Thorax: 2001;56: 4–8.

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Results

There was a greater prevalence of reported wheezing, use of asthma medications over the previous year and exertion-related shortness of breath in the severely obese participants compared with individuals who had a normal BMI. There was a trend for greater prevalence of asthma in the severely obese individuals. After adjusting for atopy, age, gender, smoking history, and family history of asthma, severe obesity was associated with an elevated odds ratio (OR) for wheezing over the previous 12 months (OR: 2.6, $P = 0.001$), a diagnosis of recent asthma (OR: 2.04, $P = 0.048$) and use of medication for asthma during the preceding year (OR: 2.83, $P = 0.005$). Notably however, severe obesity was not a risk factor for AHR (OR: 0.72, $P = 0.73$). The investigators did not observe a significant correlation between BMI and AHR. In addition, there was no difference between the severely obese participants and those of normal weight with respect to FEV_1/FVC ratio, peak expiratory flow or mid forced expiratory flows. Specifically, there was no indication that the severely obese participants had a greater degree of airways obstruction than normal weight individuals.

The investigators also made the interesting observation that compared with normal weight individuals, underweight participants had more pulmonary symptoms, abnormal pulmonary function and increased AHR.

Conclusion

The investigators concluded that although severe obesity is associated with an increased likelihood of wheezing and shortness of breath, the absence of AHR suggests that this does not reflect asthma, at least as reflected by bronchial hyper-reactivity. They speculate that wheezing in these patients may be due to anatomic changes associated with reduced resting lung volumes or other factors. They also offered the possibility that the question regarding wheezing that they posed to patients may be insufficiently specific. In light of their observations that despite the increased risk for being on asthma medication and the self-reported wheezing and asthma associated with severe obesity, these individuals did not exhibit bronchial hyper-responsiveness, the investigators concluded that these medications may be over-prescribed in severely obese patients. They further speculate that some of these medications (e.g. corticosteroids) could, in fact, exacerbate the bariatric problem.

Comment

Why was this paper selected for commentary in Sleep Medicine? As Sleep Medicine clinicians, it is likely that we care for a greater proportion of severely obese patients than most of our colleagues in other fields. We conduct a detailed medical interview with these patients focused on the issues of breathing problems in relations to sleep and will commonly obtain a history of shortness of breath and asthma. Often we have been consulted on these patients due to a suspicion of Sleep-Disordered Breathing and a history, or physiological evidence of asthma during wakefulness usually prompts more careful consideration that this may be an etiologic factor and perhaps, therapy directed toward asthma (including nocturnal asthma) is in order. The physiologic hallmark of asthma is bronchial hyper-reactivity in response to inhalation provocation with histamine or methacholme. This paper by Schachter and coworkers reminds us that a history of wheezing and/or asthma provided by severely obese patients does not necessarily reflect the presence of bronchial hyper-reactivity as defined by such challenge. Consequently, we should not assume that recent wheezing or asthma reported by a severely obese patient provides solid evidence for the presence of this disorder. This is not to say however, that such a history should be ignored. Indeed, asthma and Sleep-Disordered Breathing may co-exist and each should be investigated in an appropriate manner. This means that Sleep Medicine clinician who obtains a history of recent wheezing or asthma, particularly from a severely obese individual should investigate more completely how this diagnosis was made. If the definitive physiologic studies for asthma had not been conducted, arrangements should be made for a consultation as needed to ensure a proper evaluation in this regard, as well as for an appropriate assessment for Sleep-Disordered Breathing. The clinician should recognize that obesity increases the risk for wheezing and shortness of breath but NOT for asthma, defined by bronchial hyper-reactivity.