Impact of obesity on ventilatory function

Perran Boran,1 Gulnur Tokuc,2 Burcu Pisgin,3 Sedat Oktem,1 Zeliha Yegin,3 Ozlem Bostan3

Abstract

Objective: Although obesity was found to be associated with severe impairment of ventilation, most of the study population has been morbidly obese adults. We aimed to explore the effects of mild obesity on ventilatory function in the pediatric age group.

Methods: In a cross-sectional controlled study, 80 patients (M/F: 35/45), who were evaluated in our outpatient clinic with the complaint of excess body weight, with no history of asthma or other atopic diseases were studied and compared to a control group of 50 normal weight children controlled for age and sex. The mean age of patients was 9.7±2.5 years (7 to 15 years). Anthropometric measurements and spirometry were performed in all subjects. Forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV1) were used as measures of ventilatory function.

Results: There were no significant differences in FEV1%, FVC% and FEV1%/FVC% by study group (p > 0.05). Only three patients had obstructive abnormalities documented on their pulmonary function tests (two had moderately severe and one had mild obstructive abnormalities). No correlation was observed between pulmonary function parameters and anthropometric measurements.

Conclusion: These data demonstrate that pulmonary function test parameters of the mildly obese children were similar to those of the normal weight children. Anthropometric measurements had no significant effect on spirometric measurements in children as they did on adults.


Introduction

Childhood obesity is associated with a range of adverse consequences.1 Concerns about obese children include metabolic and physical disorders other than psychosocial stress. Abnormalities of the respiratory function have been observed in many studies.2-4

Several mechanisms have been proposed on the possible effects of obesity on pulmonary function. The most common abnormalities reported are reduced expiratory reserve volume and functional residual capacity due to reduced chest wall and lung compliance and increased respiratory resistance.5,6 It is also believed that increased pulmonary blood volume leads to congestion resulting in thickening of the airway wall; thus reducing airway size.7

Although morbid obesity was found to be associated with severe impairment of ventilation, studies on the effects of mild obesity on ventilatory function are limited.3

1. MD. Pediatrician, 2nd Clinic of Pediatrics, Dr. Lutfi Kirdar Kartal Research and Training Hospital, Istanbul, Turkey.
2. MD. Associate professor, 2nd Clinic of Pediatrics, Dr. Lutfi Kirdar Kartal Research and Training Hospital, Istanbul, Turkey.
3. MD. Resident, 2nd Clinic of Pediatrics, Dr. Lutfi Kirdar Kartal Research and Training Hospital, Istanbul, Turkey.

Manuscript received Jul 31 2006, accepted for publication Dec 13 2006.

It was hypothesized that obesity can have adverse effects on ventilatory function even in mildly obese children. The purpose of this article is to explore the effects of simple obesity on ventilatory function.

Methods

We carried out a cross-sectional controlled trial in 100 patients who were admitted to Dr. Lutfi Kirdar Kartal Training and Research Hospital, pediatric outpatient clinic, with the complaint of excess body weight, and compared to a control group of normal weight children of similar ages, between November 2004 and May 2005. The control group was composed of healthy children who have attended our outpatient clinic for their routine checkups, vaccines, nutritional assessments and who have normal physical examination.

Based on information from previous studies, a sample size of 50 children per group was calculated to be adequate to detect a difference.

In this study an obese subject was defined as one whose BMI was above the 95th percentile according to sex- and age-specific BMI reference range using the new charts provided by the Centers for Disease Control and Prevention. Those with obesity secondary to an organic condition (one patient had Hashimoto’s thyroiditis); having a chronic cardiorespiratory or neuromuscular problem or history of asthma and other atopic diseases were excluded from the study. The remaining 80 patients (M/F: 35/45; mean age 9.7±2.5 years) with obesity were enrolled into the study. They were compared with 50 healthy normal weight children (mean age 9.2±2.08). The male/ female ratio of the obese and normal weight children were 35/45 and 20/30, respectively.

A questionnaire was administered by the investigators to determine risk factors including daily TV viewing time, eating habits, daily physical activities, and family history of obesity. Parents were asked about the child’s snoring, difficulty breathing, observed apnea, cyanosis, struggling to breathe, shaking the child to “make him or her breathe,” watching the child sleep, afraid of apnea, the frequency and loudness of snoring, and daytime symptoms such as excessive daytime sleepiness to determine obstructive sleep apnea (OSA) symptoms.

An informed consent was obtained from the subjects and their parents.

Anthropometric measurements and spirometry were performed in all subjects. Height was measured to the nearest 1 cm against a wall chart, and weight was measured to the nearest 0.1 kg using an electronic digital scale. BMI was calculated as weight (kg) divided by the square of height in meters (kg/m²). Waist circumference was measured as the minimum abdominal circumference between the xiphoid process and the umbilicus. Hip circumference was measured as the maximum circumference over the buttocks. The waist-to-hip ratio (WHR) was calculated as the ratio between these two circumferences. The height and weight of patients’ parents were also measured by the same physician and BMI was calculated.

Spirometry (Spiromed- microplus M503, MAN5105, spirometer) was performed in all subjects. The best of at least three technically acceptable values for forced expiratory volume in 1 second (FEV1) and forced vital capacity (FVC) were selected. Forced vital capacity and FEV1 were used as measures of ventilatory function. The pulmonary function test results were expressed as percentages of predicted normal values. For the purpose of this study, the threshold of abnormality was identified as less than 80% of the predicted value. Obstructive airway disease was identified as a decrease in the FEV1/FVC ratio to less than 80%. The various pulmonary deficits were classified as “mild” (> 70%), “moderate” (< 70% and > 60%), “moderately severe” (< 60% and > 50%), and “severe” (< 50%). The reversibility test was applied to subjects whose FEV1/FVC ratio decreased to less than 80%. Salbutamol sulfate was used for the test and it was inhaled twice and FEV1 was measured 15 minutes later. If the difference in FEV1 before and after bronchodilator inhalation was greater than or equal to 15%, the test was accepted as positive.

The data were analyzed by SPSS software 10.0. Numerical variables were evaluated by one-sample Kolmogorov Smirnov test to assess whether they followed a normal distribution. Since the parameters were not normally distributed, a nonparametric Mann-Whitney U test was used. Medians, 1st and 3rd quartiles were computed to describe respiratory function parameters. A p value less than 0.05 was considered statistically significant. The chi-square test was used to compare sex and age distribution in both groups.

Regression models were built and the covariates were tested by the enter and forward approach. Several risk factors such as weight, body mass index, relative weight and waist-to-hip ratio were included as independent variables.

Results

Eighty children with exogenous obesity aged between 7 to 15 years (mean age 9.7±2.5 years) were enrolled and compared with 50 healthy normal weight children (mean age 9.2±2.08 years). The male/ female ratio of the obese and normal weight children were 35/45 and 20/30, respectively. Demographic characteristics and obesity-associated risk factors are given in Table 1.
The children in the two groups were comparable for a number of baseline characteristics, including age, sex, and child feeding practices, parental obesity, passive cigarette smoking, OSA symptoms and performance of physical activity, except for television viewing. Significantly more obese subjects are reported to watch television more than one hour a day (p < 0.05).

Anthropometric measurements of the obese and control groups are given in Table 2.

The mean values of weight, relative weight, BMI, and WHR were significantly higher in the obese group, as expected (p < 0.01). There were no significant differences in age and sex by study group (p = 0.888).

Mildly obese subjects compared to nonobese subjects did not differ in any of the lung function measurements (Table 3).

Three patients in the obese group had obstructive abnormalities documented on their pulmonary function tests (two had moderately severe and one had mild obstructive abnormality). The reversibility test was positive in these three patients. These three patients had no asthma symptoms such as dyspnea, wheezing, chronic cough or previous history of atopy.

According to regression analyses, anthropometric measurements had no significant effect on FEV1% (p = 0.3), FVC% (p = 0.545), and FEV1/FVC% (p = 0.869).

**Table 1** - Baseline characteristics of children enrolled in the obese and control groups

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Obese</th>
<th>Control</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>9.7±2.5</td>
<td>9.2±2.08</td>
<td>0.869</td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>35/45</td>
<td>20/30</td>
<td>0.807</td>
</tr>
<tr>
<td>Parental BMI &gt; 30</td>
<td>20/80 (25%)</td>
<td>13/50 (26%)</td>
<td>0.313</td>
</tr>
<tr>
<td>Passive cigarette smoking</td>
<td>53/80 (66.2%)</td>
<td>32/50 (64%)</td>
<td>0.725</td>
</tr>
<tr>
<td>Physical activity &lt; 1 h/day</td>
<td>44/80 (55%)</td>
<td>26/50 (52%)</td>
<td>0.513</td>
</tr>
<tr>
<td>Fast food eating</td>
<td>66/80 (82.5%)</td>
<td>40/50 (80%)</td>
<td>0.625</td>
</tr>
<tr>
<td>Television viewing &gt; 1 h/day</td>
<td>76/80 (95%)</td>
<td>40/50 (80%)</td>
<td>0.019</td>
</tr>
<tr>
<td>Breastfeeding &lt; 6 months</td>
<td>32/80 (40%)</td>
<td>20/50 (40%)</td>
<td>0.840</td>
</tr>
<tr>
<td>OSA symptoms</td>
<td>8/72 (11%)</td>
<td>5/50 (10%)</td>
<td>0.766</td>
</tr>
</tbody>
</table>

BMI = body mass index; OSA = obstructive sleep apnea.

**Discussion**

Many studies have demonstrated an association between obesity and ventilatory abnormalities in adults. However, investigations into this issue in childhood are limited and studies conducted to date have yielded conflicting results. Many have focused on extreme levels of obesity, or have used a small sample size.

Chaussain et al., in their study of 39 obese children, reported that lung compliance and resistance reflected as vital capacity and residual volume were similar to those of the control group. Bosio et al., in their study of 23 obese children, also found lung volumes to be within the normal range. Consistent with these studies, our results revealed that FEV1 %, FVC % and FEV1%/FVC% were similar to those of the control group.

Similar studies in children confirm reduced functional residual capacity and static lung volumes. Mallory et al. found that 3 out of 17 obese patients had restrictive and 8 out of 17 had obstructive changes in pulmonary function. Inselman et al. and Li et al. found reductions in diffusing lung capacity to be common among the obese children they studied. They suggested that reductions in diffusing lung capacity seen in children may reflect structural changes in the interstitium of the lung, resulting in decreased alveolar surface area.

One possible explanation for these conflicting results may be that most of the studies have focused on extreme levels of
obesity, or have used small sample sizes without a control group. It is also possible that conventional respiratory function tests are only mildly affected except in extreme cases and that individuals with different levels of obesity will exhibit a different response. Ray et al. emphasized that total lung capacity and vital capacity may be reduced only in extreme obesity.\textsuperscript{16}

Although abnormalities of the respiratory function are a common finding in adult obesity, we can not draw any conclusions from adult studies since physiological function and body fat deposition are different from those of children and also there are so many confounding factors such as smoking status, and an abnormal pulmonary function test value can be considered to be caused by intrinsic lung disease or factors other than obesity.

Previous studies suggested that patterns of fat deposition are important in determining the consequences of obesity and that high WHR is inversely related to spirometry and

<table>
<thead>
<tr>
<th>Table 2 - Anthropometric measurements of obese patients and controls</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Weight (kg)</strong></td>
</tr>
<tr>
<td>-----------------</td>
</tr>
<tr>
<td>Obese</td>
</tr>
<tr>
<td>Nonobese</td>
</tr>
<tr>
<td>p</td>
</tr>
</tbody>
</table>

BMI = body mass index. Values expressed as means ± SD.

<table>
<thead>
<tr>
<th>Table 3 - Pulmonary function tests of obese and normal weight children</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Obese</strong></td>
</tr>
<tr>
<td>------------</td>
</tr>
<tr>
<td>FEV\textsubscript{1}%</td>
</tr>
<tr>
<td>IQR\textsubscript{25-75}</td>
</tr>
<tr>
<td>FVC%</td>
</tr>
<tr>
<td>IQR\textsubscript{25-75}</td>
</tr>
<tr>
<td>FEV\textsubscript{1}%/FVC%</td>
</tr>
<tr>
<td>IQR\textsubscript{25-75}</td>
</tr>
</tbody>
</table>

FEV\textsubscript{1}% = percentage predicted forced expiratory volume in 1 second; FVC\% = percentage predicted forced vital capacity; IQR = interquartile range. Data are presented as medians, 1st and 3rd interquartile ranges.
Effects of obesity on respiration – Boran P et al.

Conclusions

It is also possible that anthropometric measurements have failed to determine fat distribution accurately. Conventional anthropometric measurements have been criticized as being unreliable and insufficiently sensitive to assess intraabdominal fat. A more valid and precise measure of body fat distribution, such as measurements obtained with modern investigation methods such as CT, MRI or DEXA (dual energy X ray absorptiometry) would be preferred, but we did not want to expose patients to radiation.

Furthermore, the deposition of visceral fat is very age-dependent; in one study, visceral fat was shown to increase in men from 12.4% of body surface at age younger than 40 years to 18% after age 65. This increase was independent of obesity. By contrast, the figure was 5.4% for adolescents and adiposity for male and female children is predominantly subcutaneous which may not constitute a great health risk.

There have been reports in the literature suggesting an association between asthma and obesity. Although three patients had reversible obstructive abnormality documented on their pulmonary function tests, they had no respiratory or atopic symptoms previously and since we have not performed any provocations tests, further studies are needed to determine whether obesity causes or enhances bronchial hyperreactivity.

Our study has certain limitations. First, it was a cross-sectional study and, since measurements of the obese subjects were taken at a single point in time, they may not have accurately reflected the clinical status. Second, radiological assessment would have been helpful in this study, since it is capable of determining fat distribution more accurately than anthropometric indices.

Conclusion

In conclusion, baseline pulmonary function test parameters were not different between mildly obese and normal weight children. Anthropometric parameters had no significant effect on pulmonary function. Longitudinal studies including physiological tests are needed to explore the effects of different levels of obesity on pulmonary function in children.

Acknowledgements

The authors are grateful to Haydar Sur for the statistical analyses.

References


Correspondence:
Perran Boran
Akin sok Hatboyu Cikmazi
Ortac apt 13/12, Saskinbakkal
Istanbul, 34740 – Turkey
Tel.: +90 (532) 7127756
Fax: +90 (216) 4110877
E-mail: drperran@yahoo.com