Potential connection of dyslipidemia with BMI and associated disorders in obese children and adolescents

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Objectives:

For children and adolescents under 19 years, the prevalence of obesity has remained stable at about 17% and affects about 12.7 million children and adolescents for the past decade [7]. Body Mass Index, or BMI, is used as a screening tool for overweight or obesity. It is defined to determine body weight status in children and adolescents using age- and gender-corrected standard deviation scores for body mass index (BMI-SDS). Body Mass Index (BMI) is a person’s weight in kilograms divided by the square of height in meters. Also excess body weight (EBW) is increasingly applicable to children and adolescents (BMI 99-th percentile). BMI seems to be informative for the severity of obesity, but not for relevant risks of dyslipidemia. BMI does not measure body fat directly, but research has shown that BMI is moderately correlated with more direct measures of body fat obtained from skinfold thickness measurements [2, 3, 12]. Moreover, BMI is strongly correlated with various metabolic disorders and disease outcome consistent with these more direct measures of body fatness and health state [1, 6]. Obesity can occur at any age, even in young children and overall quality of life may be diminished. Current investigation reports are there potential connections between absolute values of lipids levels and BMI in obese children and adolescents, as well as the relationships between dyslipidemia and several associated disorders.

Methods:

In 34 children and adolescents aged 4-18 years (average age of 11.3) with obesity (BMI > 99 percentile) and excess body weight (99 percentile) the values of triglycerides (TG), total cholesterol (C), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) were measured to evaluate existence of dyslipidemia and compared these values with those of the age- and sex- specific reference group in the Lipid Research Clinics Population Studies Data Book (LRCP). The HOMA-index of insulin resistance, as well as the levels of insulin, pro-insulin and thyroid status was also evaluated. Male/female ratio was 20/14. BMI was used as a basis for weight status. To correct for age and sex, individual BMI-values were converted to Z-scores (SDS values). These references were derived using Cole’s LMS method (Cole and Green 1992), which allows BMI to convert to SD scores as follows:

\[ \text{SDS} = (X - \text{Median}) / \text{SD} \]

where X = individual BMI-value, L = Box-Cox power, M = Median, and S = coefficient of variation for individual age (t) and sex from the reference group. Results are indicated as BMI-SDS values.

Statistical analyses were performed to determine the significance of findings. All statistical analyses were performed using SPSS 11.5.2.1 (SPSS Inc., Chicago, IL). In all cases null hypothesis was rejected if p<0.05.

A total of 67.6% of the overweight children showed an abnormal lipid profile (fig. 1).

No any correlation has been found between absolute values of lipid fractions levels and BMI (r<0.02). Also there were no significant connection between dyslipidemia and gynecomastia, stealathropesia, hypothyroidism, sex, and pubertal delay (p>0.05).

Interestingly in 70.6% of investigated patients the insulin-resistance and hyperinsulinemia were found, 91.7% from which had statistically significant dyslipidemia (p<0.05) without sex predominance (fig.2). Insulin-resistance and hyperinsulinemia are positively associated with BMI-SDS>2.5.

Conclusions:

Obese children develop not only insulin resistance but also dyslipidemia as result of obesity. Recently published pediatric studies have focused on atherosclerosis as a process beginning in early childhood and related to obesity. Framingham Study data showed an increased incidence of cardiovascular events with increasing bodyweight, independent of gender. Considering these results, lipid value screening appears to be helpful at the beginning of an intervention program for obese children. Hyperinsulinemia and insulin-resistance are connected with severity of obesity (BMI-SDS>2.5) and are mostly associated with obesity in children and adolescents. We assume that there are marginal values of lipids’ levels above which the BMI doesn’t have interaction on them or other regulator mechanisms are triggered. The mechanism of change in the lipid profile could be hyperinsulinemia causes hepatic VLDL-synthesis and leads to increased TG- and LDL-C-levels. The lack of the effect of insulin on lipoproteinipase in peripheral tissues could then result in an increase in TG and LDL-C. In addition, hyperinsulinemia and insulin-resistance in children leads to further alterations in fatty acid oxidation and concentrations [8]. Other metabolic factors, such as an increase in levels of apolipoprotein B, homocysteine, and C-reactive protein are risk factors for atherosclerotic coronary heart disease [4, 5, 9].

Despite, medical treatment of dyslipidemia in childhood is controversial and is so far only indicated for familial heterozygotic hypercholesterolemia [10, 11]. Further investigations should be done to evaluate the relationships between other associated endocrine and somatic disorders and hyperinsulinemia and/or insulin-resistance and BMI severity in obese children and adolescents.

References: