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Original Article

Obesity and secondary sexual maturity in boys

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Abstract

Background Obesity induces earlier secondary sexual maturity in girls. However, results of studies in boys have been inconclusive.

Objective To assess for an association between obesity and sexual maturity in boys.

Methods This was a prospective cohort study on 133 obese and 133 matched-for-age, non-obese, prepubertal boys, aged 9 to 10 years. They were observed every 4 months for a two year period. Obesity was defined as the BMI ≥the 95th percentile, according to the *Centers for Disease Control* 2000 growth charts. Secondary sexual maturity was defined as testicular volume of ≥4 mL and Tanner staging of pubic hair of ≥P2. Two trained nurses and a pediatric resident collected the data.

Results By the end of the study period, 106 (79.7%) obese boys had attained a testicular volume of 4 mL or more, vs. 85 (63.9%) non-obese boys (RR 1.78; 95%CI 1.19 to 2.67; P=0.004). Furthermore, 81 (60.9%) obese boys attained pubic hair states of Tanner stage P2 vs. 37 (27.8%) non-obese boys (RR 1.85 95%CI 1.46 to 2.34; P<0.001). The mean durations for achieving testicular volume of ≥4 mL were 21.25 (95%CI 20.75 to 21.74) months in obese boys and 22.26 (95%CI 21.80 to 22.72) months in non-obese boys (P=0.007). The mean durations for achieving Tanner stage P2 were 17.04 (95%CI 16.44 to 17.63) months in obese boys and 20.87 (95%CI 20.05 to 21.68) months in non-obese boys.

Conclusion Obesity is associated with earlier onset of sexual maturity in boys. **[Paediatr Indones. 2013;53:283-6.]**

Keywords: *obesity, boys, secondary sexual maturity, testicular volume*

Obesity has become a health problem around the world. The World Health Organization stated that obesity is a global epidemic that needs to be solved. The prevalence of obesity in children has been increasing in both developed and developing countries.[1]

The problem of obesity is a serious concern, partly because of the traditional societal perception that fat equates to health, while in reality, obesity is the cause of many health problems. Approximately 75-90% of heart disease cases were related to dyslipidemia, hypertension, diabetes mellitus, smoking, low activity and obesity.[2] A study in Denmark concluded that a higher body mass index (BMI) in childhood leads to a higher risk for acquiring coronary heart disease in adulthood.[3]

A study by Wang found a positive association between obesity and earlier sexual maturity in girls (OR 2.0; 95%CI 1.1-3.5), but a negative one in boys (OR 0.4; 95%CI 0.2-0.8).[4] In addition, earlier sexual maturity was observed in Hispanic children compared to Caucasian children.[4] Most studies on an association

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between obesity and sexual maturity use a cross-sectional design, whereas longitudinal studies have rarely been used, especially in Indonesia.[5]

The objective of this study was to assess for an association between obesity and sexual maturity in boys, employing a two-year, follow-up study.

Methods

The design of this study was a prospective cohort. Subjects were elementary school boys from 32 schools in Yogyakarta and Sleman.

Obesity was defined as a BMI ≥the 95th percentile of the Centers for Disease Control (CDC) 2000 reference population. Sexual maturity was assessed by Tanner staging and testicular volume was measured by orchidometer. The boys were regarded as entering sexual maturity if their testicular volume was ≥4 mL and/or their pubic hair growth stage was of ≥stage P2.

At the beginning of the study, all subjects were prepubertal and aged 9 to 10 years. Obese subjects were compared to non-obese subjects, i.e., BMI < 85th percentile using the same standard population, and matched for age. Subjects were observed at baseline, and every 4 months for a 2 year period (2006-2008). We excluded boys with short stature because of the possibilty of having pathological obesity, such as Prader-Willi and Cohen syndromes.

Physical examination and measurements of testicular volume were performed by two trained nurses and a pediatric resident. Interobserver agreement between the three examiners was good, with Kappa scores for pubic hair examination between the first and second examiner, the second and third examiner, and the first and third examiner of 0.80, 0.66 and 0.76, respectively. For testicular volume measurements, the Kappa scores were 0.89, 0.84 and 0.90, respectively.

Data was processed using survival analysis and Chi-square test. Level of significance was defined as $P < 0.05$.

Results

We screened 1,827 boys for obesity from 16 elementary schools in Yogyakarta and 16 elementary schools in Sleman, yielding 133 (7.3%) obese, 189 (10.4%) overweight, 1,315 (72.2%) normal and 184 (10.1%) underweight boys. All 133 obese boys were included in our study, and 133 non-obese boys matched-for-age were selected as controls. Baseline characteristics of the study subjects are shown in **Table 1**.

At the end of study, 118 (54.4%) subjects had reached pubic hair maturity (Tanner stage of ≥P2), 81 (60.9%) obese boys and 37 (27.8%) non-obese boys. After two years of follow-up, the relative risk for pubic hair maturity in obese group was 1.85 (95%CI 1.46-2.34, P<0.001).

The Kaplan-Meier curve in **Figure 1** shows the progression of pubic hair maturity. The mean pubic hair maturity in the obese group began after 17.04 (SD 2.74) months of the study (95%CI 16.44 to 17.63), while that of the non-obese group began after 20.87 (SD 2.52) months of the study (95%CI 20.05 to 21.68, P<0.001). This was correlated to a mean age for pubic hair maturity of 10.70 years in the obese group and 11.01 years in the non-obese group.

Table 1. Baseline characteristics of the study subjects

Characteristics	Obese group n=133		Non-obese group n=133		P value
Mean age (SD), years	9.28	(0.42)	9.28	(0.42)	0.99
Mean weight (SD), kg	41.12	(1.35)	32.46	(1.45)	<0.001
Mean height (SD), cm	136.21	(6.72)	137.90	(1.76)	0.03
Mean BMI (SD), kg/m^2	22.06	(1.03)	17.12	(0.76)	<0.001
Mean number of family members (SD), n	4.09	(0.80)	4.48	(0.92)	<0.001
Pubic hair P1 at baseline (%), n	133	(100)	133	(100)	-
Testicular volume ≤2 mL at baseline (%), n	133	(100)	131	(98.5)*	0.16**
Positive family history of obesity (%), n	94	(70.7)	82	(61.7)	0.12

* Two non-obese subjects had testicular volume of 3 mL at baseline

**Fischer's exact test

At the end of study, 191 (71.8%) children had reached a testicular volume of ≥4 mL, 106 (79.7%) obese boys and 85 (63.9%) non-obese boys. The relative risk for accelerated testicular volume ≥4 mL in obese group was 1.78 (95%CI 1.19-2.67, P= 0.004).

The Kaplan-Meier curve in **Figure 2** shows the progression of children achieving testicular maturity. The mean testicular volume of ≥4 ml in the obese group began after 21.25 (SD 2.61) months of the study (95%CI 20.75 to 21.74), while that of the non-obese group began after 22.26 (SD 2.18) months of the study (95%CI 21.80 to 22.72, P=0.007). This was correlated to a mean age for testicular maturity of 11.05 years in the obese group and 11.12 years in the non-obese group.

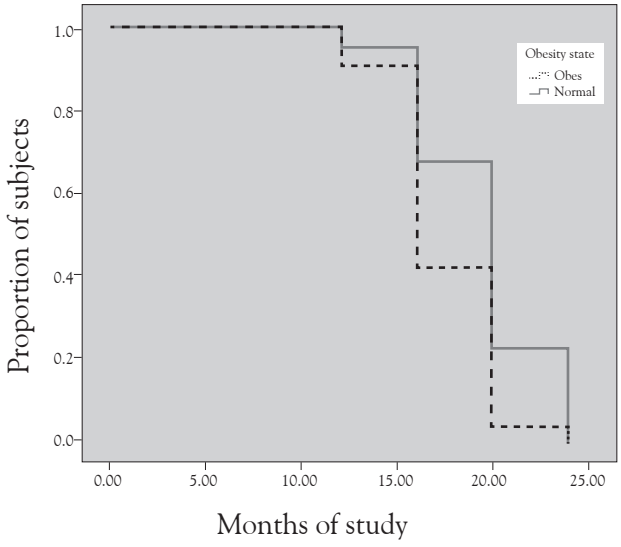

Figure 1. Kaplan-Meier curve for pubic hair maturity (Tanner stage ≥P2)

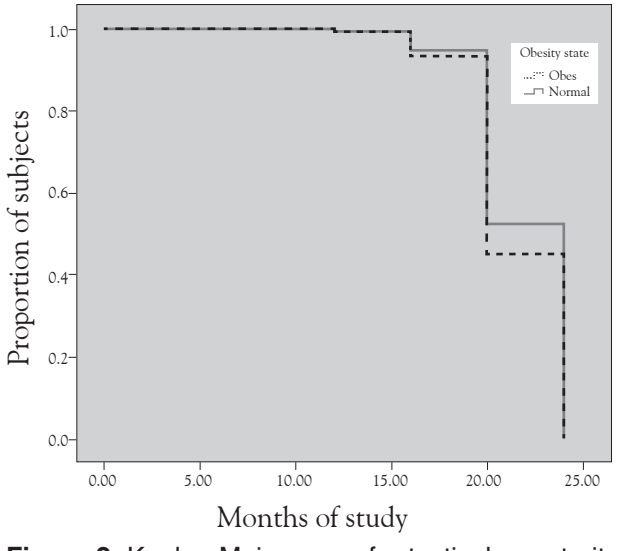

Figure 2. Kaplan-Meier curve for testicular maturity (≥4 mL)

Discussion

The age range of our study subjects at the time of recruitment was chosen with the assumption that within two years of observation, some of the boys would achieve secondary sexual maturity. It has been reported that secondary sexual characteristics in boys begin at the age of 10.5 to 12 years.[5] Observations were perfomed every four months, in order to not miss detecting changes in the sexual maturity of our subjects. [6]

At baseline, all boys had pubic hair growth of Tanner stage P1, and almost all boys had testicular volume of 1 mL. According to Biro *et al.*, characteristics of sexual maturity in prepubertal boys are pubic hair growth at stage P1 and testicular volume of ≤3 mL. Testicular volume of 4 ml is an indicator of pubertal onset.[7]

In our study, the obese boys reached sexual maturity earlier than the non-obese boys. Similarly, Lee *et al.* observed that at the age of 11.5 years, more boys with high BMI had entered puberty than boys with low BMI.[8]

We also found that obesity was a risk factor for accelerated testicular maturity. Similarly, Lee *et al.* found that boys with high BMI (overweight or obese) had a higher risk for early puberty compared to boys with low BMI, with a relative risk of 2.63 (95%CI 1.05-6.61, P=0.04).[8] In contrast, Wang observed a negative association between sexual maturity in boys, as obese boys entered sexual maturity later than non-obese boys in their study, (OR=0.4; 95%CI 0.2 to 0.8).[4]

Wirawan *et al.* found no significant statistical differences in the development of secondary sexual characteristics between well-nourished and undernourished male students (based on BMI z-score).[9] However, Hartoyo *et al.* showed that well-nourished boys had earlier average age of spermache, appearance of secondary sexual characteristics and higher sexual maturity rating (P<0.05).[10]

One possible explanation for the different results of these studies may be the study designs. The three former studies[8-10] used a cross-sectional design, while we used a prospective cohort design. We expect the observation of secondary sexual maturity to be more accurate in our study because it was done repeatedly.

A limitation of our study was that the assessment of nutritional status was conducted only at the beginning of the study, so changes in nutritional status over the two year observational period were not detected. We also did not take into account other risk factors associated with accelerated secondary sexual maturity in boys, e.g., living and eating habits. The hormones associated with puberty were also not evaluated. In conclusion, we found that obesity is associated with earlier onset of sexual maturity in boys.

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