Onset of Puberty in Relation to Obesity

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The process of pubertal development and the factors affecting it are only partly understood even today. Owing to the rising epidemic of childhood obesity, there have been a number of studies investigating the role of body composition, and the rate and timing of puberty. There is evidence now that increased height and body mass index (BMI) of children, prior to puberty, result in an earlier onset of puberty [1,2].

Rapid weight gain in early life has been linked to advanced puberty in both sexes, more so amongst girls [3]. Early onset of thelarche has been reported in girls with high BMI at the age of three years, and in those with a rapid elevation of BMI from three years of age to the age of entry into the first grade [4]. The rapid catch-up growth in children born small-for-gestational age [5] with associated decreased insulin sensitivity, and increased IGF-1 levels, also results in early onset of pubarche for the same reason.

A definite association exists between increasing BMI and earlier pubertal development in girls, and the study [6] published in this issue of *Indian Pediatrics* also confirms this association. Majority of studies suggest early puberty and voice cracking in obese boys, but some studies have contradictory results. Till recently, little was known regarding genes regulating puberty. Studies have now identified gene loci [*myocardin-like 2 (MKL2)* for male sexual development, menarche locus linking earlier puberty with reduced pubertal growth, and loci for short stature in boys and girls] that may explain the factors influencing obesity and pubertal changes [7]. BMI-increasing alleles in girls correlated well with earlier breast development but in boys they are associated with early sexual development in some and delayed sexual development in others. This could explain the conflicting results of studies analyzing pre-pubertal obesity and onset of puberty in boys. Though the mechanisms regulating pubertal onset in males and females may be similar to a large extent, the relationship in boys may be complex requiring further genetic studies.

Adiposity has been proposed as a metabolic gatekeeper of central pubertal initiation but the postulation that central activation of GnRH-gonadotropin axis in obese girls may be the cause for premature thelarche has not been proven. The peripheral aromatization of the vast adipose tissue androgens to estrogens [8], the insulin-induced reductions of sex hormone binding globulin (SHBG) which increases bioavailability of sex steroids including estradiol [9], and the hyperinsulinemia in obese girls contribute to early onset of thelarche. However, the time from thelarche to menarche may be delayed in obese girls suggesting that the increase in estrogen in obesity may not be gonadotropin-dependent [10], and the isolated menstrual bleeds may represent non-ovulatory bleeding.

Obesity increases pubertal insulin resistance, especially in girls [11]. This and the resultant hyperinsulinemia may advance pubertal maturation in these children. The fact that African-American children, who are genetically prone to insulin resistance, attain pubertal milestones earlier than their peers [12], highlights the affect of insulin resistance on puberty.

As mentioned earlier, we are still in the dark regarding various aspects of the influence of obesity on pubertal development. Several questions remain unanswered. More research is needed to clearly delineate the effects of excess adiposity on pubertal development in boys. Whether gonadotropin-dependent or independent mechanisms underlie early thelarche in obese girls remains to be established. Also implications of hyperandrogenemia during early puberty and their role in advancing puberty need to be studied.

Developing screening tools and strategies for preventive and curative management of obesity-related reproductive problems in adolescents will need a clearer understanding of the complex etiopathogenesis of the condition in both sexes.

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