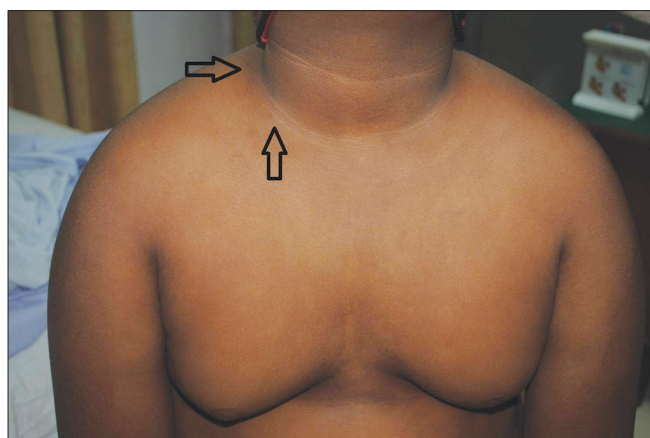


## Precocious pubarche in a young boy—unusual etiology

Sir,

Puberty is a complex physiological change in children leading to sexual maturation and reproductive capability. Precocious pubarche is defined as the appearance of pubic hair before age 8 years in girls and 9 years in boys. Though considered to be a benign entity previously, recent reports confirm a close link between precocious pubarche and metabolic syndrome in future.<sup>[1]</sup> Obesity is characterized by precocity in girls and delayed puberty in boys. Obesity leads to a state of hypogonadotropic hypogonadism in boys due to feedback effects of estrogen excess derived from peripheral aromatization of testosterone.<sup>[2]</sup> Obesity and metabolic syndrome leading to pseudoprecocity is rarely seen in boys.

An 8 1/2-year-old boy was brought by the parents after noticing progressive development of pubic hair for over 2 months. The boy is a product of non-consanguineous marriage and was delivered at term with birth weight of 3.2 kg. The motor milestones, speech, and language development were normal in the patient. The boy was obese throughout childhood and the parents denied noticing any phase of rapid growth. The boy gave no symptoms to suggest neurological, systemic or syndromic illness, and the family history was not contributory. Anthropometry revealed height 137 cm (75<sup>th</sup> to 95<sup>th</sup> centile), weight 65 kg (>97<sup>th</sup> centile), body mass index (BMI) 34.1 kg/m<sup>2</sup>, BMI for age >95<sup>th</sup> centile, abdominal circumference 93 cm, hip circumference 97 cm, and waist hip ratio 0.95. General examination revealed normal body proportions, acanthosis nigricans, and bilateral lipomastia [Figure 1]. Pubertal staging according to Tanner was A1P2G1, with testicular volume of 4 ml and stretched penile length of 4.2 cm



**Figure 1:** Bilateral lipomastia with acanthosis nigricans

[Figure 2]. Bone age according to Greulich and Pyle was 10 years.

His basal and stimulated LH were 0.01 and 1.3 IU/L, respectively. He had normal stimulated FSH 1.2 IU/L, total testosterone 69 ng/dl, and elevated DHEAS 92 µg/dl (normal 12–42 µg/dl), and estradiol 0.4 ng/dl (normal 0.1–0.4 ng/dl). His thyroid profile, ACTH stimulated 17-hydroxy progesterone, prolactin, and hematological parameters were normal. Fasting blood glucose was 117 mg/dl, fasting serum insulin 21 µU/ml, HOMA-IR 6.2, HbA1c 6.1%, total cholesterol 196 mg/dl, LDL cholesterol 167 mg/dl, triglycerides 225 mg/dl, and HDL cholesterol 43 mg/dl. Neuroimaging did not reveal any abnormality in pituitary and sella regions. He was diagnosed as a case of benign precocious pubarche with metabolic syndrome and severe insulin resistance. He was advised diet and lifestyle measures for diabetes and started on metformin 500 mg daily.

Excess adiposity affects pubertal development and progression. Our patient presented with precocious pubarche due to obesity and metabolic syndrome. Previous reports suggest that obesity in boys leads to delayed puberty rather than precocity.<sup>[3]</sup> Obesity leads to a form of hypogonadotropic hypogonadism by feedback inhibition of excess estrogen due to peripheral aromatization of testosterone.<sup>[2,3]</sup> Obesity in boys results in higher testicular volume and elevated adrenal steroids. Obesity, premature adrenarche, and elevated DHEAS could have lead to bone age advancement independently in our case as shown in a recent study.<sup>[4]</sup> Metabolic syndrome and Type 2 diabetes in children are nightmare to treat due to lifestyle and behavioral problems in children. Our patient had all the features of metabolic syndrome and we advised lifestyle measures along with metformin. To conclude, we report a case of pseudoprecocity in a young boy due to metabolic syndrome.



**Figure 2:** Precocious pubarche

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