Effect of body mass index on peak growth hormone in children with short stature

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Introduction: Growth hormone (GH) is secreted by the anterior pituitary gland in a pulsatile manner under the regulation of growth hormone releasing hormone (GHRH), somatostatin, and GH-releasing peptide (ghrelin). Obesity attenuates both spontaneous GH secretion and GH response to exercise. The decrease in spontaneous 24-h GH secretion in obesity has been attributed to a diminished pulsatile GH release and a shorter half-life of endogenous GH.

Aim: To determine the impact of body mass index (BMI) on Results of GH stimulation testing in children with short stature.

Subjects and Methods: Subjects included 546 GH naive children with short stature. They were subjected to history, anthropometric assessment and Tanner pubertal staging. BMI and height standard deviation scores (SDSs) were calculated using National Child Health Statistics 2000 standards. They underwent GH stimulation testing using insulin and clonidine, without sex steroid pretreatment; and bone age assessment. Children with known genetic syndromes, congenital heart disease, renal failure, chronic hemolytic anemia, neoplasms, other endocrinopathies or receiving medications that may affect endogenous GH secretion were excluded.

Results: Mean BMI SDS was -0.17 ± 2.1. Median peak GH level by insulin provocation was 5.6 μg/liter. On univariate analysis, BMI SDS was significantly and negatively associated with peak GH by clonidine (r = -0.23; P < 0.0001) and insulin (r = -0.13; P < 0.003). Height, BMI SDS, bone age and Predicted adult height SDSs all were significantly associated with peak GH by insulin. Univariate analysis showed significant positive correlation between age and peak GH level by both clonidine and insulin (P = 0.60 and 0.51, respectively). It also showed significant positive correlation between height SDS, bone age SDS and peak GH by clonidine provocation. Significant negative correlation between BMI SDS and peak GH level by clonidine. Similar finding with peak GH level by insulin. Univariate association between BMI SDS and peak GH by insulin provocation was stronger in pubertal children (prepubertal, r = -0.06, P = 0.35, pubertal r = -0.19, P = 0.002) and equivocal between the both pubertal and prepubertal children by clonidine provocation (prepubertal, r = -0.2, P = 0.002, pubertal r = -0.2, P = 0.002 ). Peak GH by both insulin and clonidine provocation was highest in children with BMI SDS less than -1.

Conclusion: Long term nutritional status presented by BMI affect peak GH level. GH levels response to provocative test decreases with increased BMI SDS. This relationship between BMI and peak GH is not unique to obesity but rather persists in the normal and underweight pediatric population.

Diagnostic and prognostic value of lactate clearance in pediatric patients with sepsis and septic shock

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Background: Sepsis is a systemic inflammatory response syndrome caused by infectious etiology. A lactate level rise is a