

Acromegaly in an Infant

In discussing this case of a 21-month-old girl with excessive levels of growth hormone (GH; 135 ng/mL), prolactin (Prl; 370 ng/mL), and insulin-like growth factor-1 (IGF-1; 1,540 ng/mL), whose height was 97.6 cm (+4.4 SD) and whose head circumference was 55 cm (+5.5 SD), the authors briefly reviewed 22 cases of acromegaly reported in childhood. The majority had rapid linear growth, coarse facial features, and enlarged hands and feet; these are symptoms comparable to the findings that are discussed in the article. Interestingly, the 21-month-old girl had rapid head growth that preceded the significant rapid body growth. The authors postulate that the macrocephaly occurred because of rapid brain growth.

After a macroadenoma was removed from the suprasellar area, the GH and IGF-1 levels fell into the low normal range for preadolescent children. With pharmacologic testing, GH concentrations did not increase beyond 4 ng/mL, and Prl levels remained significantly elevated. GH-producing cells, but no Prl-producing cells, were observed under the microscope, util-

izing immunologic techniques. The integrated GH concentration remained relatively stable overnight (~ 2 ng/mL), and peak GH concentrations did not exceed 3 ng/mL. The authors profess perplexity because this patient continued to grow at 6 cm/y over the next 2 years. Three possible explanations are offered: (1) hyperinsulinemia, which they subsequently exclude on the basis of insulin levels found during performance of a glucose tolerance test; (2) the continuing secretion of low levels of GH overnight, also discounted because the integrated GH value reportedly is lower than values obtained in control subjects; and (3) the elevated Prl levels, which contributed to the normal IGF-1 levels and the sustained growth. Several references are cited to support the final hypothesis.

In reviewing the literature, the authors note that hyperprolactinemia occurred in 12 of 15 pediatric cases. In seven cases where the tumor was examined by immunohistochemical techniques, both Prl and GH were present. In contrast, only GH was found in the case under discussion. The authors postulate, therefore, that dis-

ruption of the inhibitory centers and/or tracts accounted for the hyperprolactinemia in this patient.

Blumberg DL, Sklar CA, David R, et al. *Pediatrics* 1989;83:998-1002.

Editor's comment—*This article provides stimulating reading for pediatric endocrinologists. It updates the count of children reported with acromegaly and the immune histochemical findings in the pituitary of these children. It also raises again the question regarding the capability of Prl to increase IGF-1 levels. The postoperative Prl levels in this girl were 30 to 120 ng/mL. Previously, Clemmons et al (J Clin Endocrinol Metab 1981;52:731) reported Prl levels greater than 100 ng/mL associated with normal adult IGF-1 levels in 20 GH-deficient patients. Very possibly, we as clinicians do not pay adequate attention to the role that increased Prl levels may play in producing normal IGF-1 levels in GH-deficient patients. All "suspect" GH-deficient patients whose IGF-1 is not in the GH-deficient range should be screened for high Prl.*

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