Genetic Steroid Disorders: Chapter 3J. Aromatase Deficiency and Aromatase Excess

Jonathan F. Russell, Jenise C. Wong, Melvin M. Grumbach

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Aromatase is a cytochrome P450 enzyme that catalyzes a critical step in the conversion of androgens (C19 steroids) to estrogens (C18 steroids). Mutations in the aromatase gene (CYP19A1) can result in a lack of aromatase activity and, as a consequence, impairment in estrogen biosynthesis. The functional consequences of this deficiency begin in utero owing to the inability of the placenta to convert androgens to estrogens. This disorder is termed aromatase deficiency (AD). Upon the initial identification of patients with AD, certain clinical findings, such as virilization of the pregnant mother and 46,XX fetus, were easily understood based on the known effects of placental aromatase on the conversion of C19 androgens and androgen precursors to estrogens. However, the discovery that the lack of estrogens in both females and males with AD leads to the absence of a pubertal growth spurt, delayed bone age, delayed epiphyseal fusion, and decreased accrual of bone mass in the male (not only in the female) has transformed our understanding of the role of estrogen on the male skeleton. It is now established that in both females and males, estrogen induces the pubertal growth spurt and mediates epiphyseal fusion. In this chapter we summarize the clinical characteristics, pathophysiology, differential diagnosis, and treatment of human AD. The implications of AD for the clinical use of aromatase inhibitors are reviewed.

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