Genetic Steroid Disorders: Chapter 3K. 46,XY DSD due to 17β-Hydroxysteroid Dehydrogenase 3 Deficiency

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17β-hydroxysteroid dehydrogenase 3 deficiency (17β-HSD3) consists of a defect in the last phase of steroidogenesis, in which androstenedione is converted into testosterone and estrone into estradiol. Patients present female-like or with ambiguous genitalia at birth and most affected males are raised as females. Virilization in subjects with 17β-HSD3 deficiency occurs at the time of puberty and almost half change to be males. Maintenance of the testes in patients raised male is safe and recommended, except when the testes cannot be positioned inside the scrotum. The phenotype of 46,XY disorders of sex development (DSD) owing to 17β-HSD3 deficiency is extremely variable and is clinically indistinguishable from other causes of 46,XY DSD such as partial androgen insensitivity syndrome and 5α-reductase 2 deficiency. Laboratory diagnosis is based on elevated serum levels of androstenedione and estrone and low levels of testosterone and estradiol, resulting in elevated androstenedione:testosterone and estrone:estradiol ratios, indicating an impairment of the conversion of 17-keto into 17-hydroxysteroids. The disorder is due to homozygous or compound heterozygous mutations in the HSD17B3 gene that encodes the 17β-HSD3 isoenzyme. Molecular genetic testing confirms the diagnosis and provides the orientation for genetic counseling. Our proposal in this article is to review the reported and our own cases of 17β-HSD3 deficiency.

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