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JOURNAL ARTICLE

Male hypogonadism due to nontumorous hyperestrogenism

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Male hypogonadism due to the nontumorous production of estrogen was studied in a patient with gynecomastia and bilateral small testicles. Both the gynecomastia and the decrease in testicular size developed in the 5-year period before presentation. Peripheral serum concentrations of testosterone were in the low to low-normal range, while those of 17 beta-estradiol (E2) were significantly elevated, as were the urinary concentrations of total estrogen. Steroid hormone concentrations were measured in the left and right spermatic veins and the left and right adrenal veins in the basal state, and after stimulation with GnRH and ACTH. Spermatic vein concentrations of E2 were 3 to 20 times higher than concentrations previously reported in normal males. Spermatic vein concentrations of testosterone were normal. The spermatic vein concentrations of androstenedione were approximately three times higher than the mean concentration of androstenedione previously reported in the spermatic vein of normal males. The concentrations of E2 and androstenedione in the adrenal veins were also significantly elevated when compared to the concentrations previously reported in normal subjects. The authors postulate that the hyperestrogenism in this patient was due to increased aromatization of the precursor substrates, testosterone in the testes, and androstenedione in the adrenals to E2 and E1 in the testes and adrenals, respectively. Alternatively, an increased abundance or activity of the 17 beta-hydroxysteroid dehydrogenase isoenzyme which converts estrone (E1) to E2 or a relative deficiency of the 17 beta-hydroxysteroid dehydrogenase isoenzyme, which converts androstenedione to testosterone, could theoretically account for the reported abnormalities.

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