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JOURNAL ARTICLE

Pituitary-testicular function of prostatic cancer patients during treatment with a gonadotropin-releasing hormone agonist analog. II. Endocrinology and histology of the testis

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Endogenous testosterone (T), LH and FSH receptors, and in vitro production of cyclic adenosine-3':5'-monophosphate (cAMP), T and some of its steroid precursors were measured in testicular tissue obtained at orchiectomy from seven prostatic cancer patients treated for 6 months with a potent gonadotropin-releasing hormone (GnRH) agonist analog (buserelin, Hoechst, 600 micrograms 3 times a day intranasally). In addition, histologic and morphometric studies were carried out on the testicular tissue. Testicular tissue from age-matched prostatic cancer patients (n = 14), whose first therapy was orchiectomy, served as controls. The peptide treatment decreased intratesticular T by 95% (P less than 0.01) and FSH receptors by 57% (P less than 0.01), but had no effect on LH receptors. The in vitro production of T decreased by 94% (P less than 0.01), but that of cAMP was unaffected. Besides T, the in vitro production of testicular 17-hydroxyprogesterone (17-OHP-4), androstenedione and 5 alpha-dihydrotestosterone (5 alpha-DHT) dropped by 71 to 90% (P less than 0.01 to 0.05) during buserelin treatment, but those of pregnenolone, progesterone and dehydroepiandrosterone (DHEA) were not affected. Histologic studies revealed considerable variation in the seminiferous epithelium of the control group, but spermatogenesis was highly suppressed in nearly all of the buserelin-treated group. The number of Sertoli cells was unaffected, but tubular diameters were reduced (P less than 0.05) by buserelin treatment. Leydig cells appeared dedifferentiated in this group, although their number per testis was not altered. These data indicate that gonadotropin suppression by GnRH agonist most likely affects testicular steroidogenesis by inhibiting 3 beta-hydroxysteroid dehydrogenase and a step(s) prior to pregnenolone formation. The treatment does not impair testicular LH binding or cAMP production, but clearly suppresses FSH receptors. Spermatogenesis in general is suppressed but with considerable variation.

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