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JOURNAL ARTICLE

Prevalent decrease of the EGF content in the periurethral zone of BPH tissue induced by treatment with finasteride or flutamide

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The aim of the present investigation is to verify whether treatment with Finasteride or Flutamide influences the regional distribution of testosterone (T), dihydrotestosterone (DHT), and epidermal growth factor (EGF) in benign prostatic hyperplasia (BPH) tissue. Thirty seven BPH patients were studied: 15 untreated, 9 treated with Flutamide (750 mg/day for 2 months), and 13 treated with Finasteride (5 mg/day for 3 months). Testosterone and DHT were evaluated by radioimmunoassay (RIA) after purification of the extracts on celite columns, and EGF was evaluated by RIA after purification on Sep-pak C18 cartridges in total tissue and in periurethral, subcapsular, and intermediate zone. In the untreated group, T, DHT, and EGF of the periurethral region are higher than those of the subcapsular zone ($P < 0.01$ for T and $P < 0.001$ for DHT and EGF). In the Flutamide group, DHT is not modified, T is increased ($P = 0.045$), and EGF is decreased in total tissue ($P < 0.02$) and in the periurethral zone ($P < 0.01$). In the Finasteride group, T is increased ($P < 0.001$), and DHT and EGF are decreased ($P < 0.001$), particularly in the periurethral zone. A positive linear correlation between DHT and EGF is observed in the Finasteride and in the untreated groups. In conclusion, in BPH the production of EGF is a DHT-dependent receptor-mediated function. The reduction of this growth factor during both treatments, associated with a fall of DHT in only the Finasteride group, is particularly evident in the periurethral zone. Since Finasteride reduces prostatic volume, mainly of the periurethral zone, we can speculate that DHT is responsible, either directly or indirectly through growth factors such as EGF for the enlargement of this region and thus responsible for urinary obstruction.