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EXPRESSION OF SURVIVIN AND BCL-2 IN BENIGN PROSTATIC HYPERPLASIA TREATED WITH FINASTERIDE

Hypothesis / aims of study

It is known that 5α -reductase inhibitor, finasteride induces apoptosis and as a result decreases prostatic volume in patient with benign prostatic hyperplasia (BPH). We assessed the expression of survivin and bcl-2 in the epithelium of benign prostatic hyperplasia treated with finasteride.

Study design, materials and methods

Immunohistochemical staining for survivin, bcl-2 and ki-67 were performed in prostatic tissue from 39 patients who underwent transurethral resection of the prostate (TURP) without medication and 31 patients who underwent TURP treated with finasteride more than 3 months for BPH. Grade of immunohistochemical staining were classified by 0 (no stain), 1 (less than 25%), 2 (between 26-50%) and 3 (more than 50%). The relationships between the expression of surviving, bcl-2, ki-67 and clinicopathological characteristics were analyzed in 2 groups.

Results

Mean age was 73.03 ± 7.02 in TURP without medication group and 74.71 ± 5.99 in finasteride medication group (p<0.283). Mean prostate volume was 45.51 ± 8.78 ml and 37.23 ± 3.36 ml, in each group (p<0.001).

A statistically significant association was observed between the expression of survivin and prostate volume (p<0.001), but not serum PSA (p=0.172). The immunoreactivity of survivin (p<0.001) and bcl-2 (p=0.001) were significantly decreased in the treatment of finasteride compared with not treated with any medicine, but that of ki-67 was similar between two groups.

Interpretation of results

We suggested that finasteride may induce the apoptosis of prostatic epithelial cell in BPH by reducing the expression of survivin and bcl-2.

Concluding message

This finding may result in the reduction of prostatic volume and may lead to improvement in urinary tract symptoms.

Disclosures

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