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The Effect of 5α Reductase Inhibitor and Estrogen in Prostate Proliferation. An Experimental Study in Rats

Abstract

This was an experimental study using posttest control group design involving Wistar strain Rattus norwegicus as experimental animal. The purpose of this study was to explain the mechanism of BPH in elderly. Samples were randomly divided into 2 groups, 1- and 2-month group, each comprising 26 rats. Each group was divided further into two subgroups, one group received combined estrogen and finasteride, and the other, receiving finasteride only, served as control group. Each subgroup consisted of 13 rats. After treatment for 1 and 2 months, the prostate was removed and examined for TGF-ß1, EGF, FGF, and proliferation. Immunohistochemistry was used for examining TGF-ß1, EGF, FGF, and the examination of proliferation was carried out using graticulae. This study employed univariate analysis with 2 sample t test as TGF-ß1, EGF, and FGF had no correlation. Data analysis used in this research was univariate analysis with 2 sample t test. Analysis result showed that estrogen could reduce TGF-ß1 significantly in 1 month and 2 month groups (p < 0.05) and estrogen also stimulated significant increase of EGF in 2 month groups (p < 0.05). Estrogen also increased proliferation significantly in both 1 and 2 month groups (p < 0.05) but estrogen did not increase FGF significantly in both groups. Multiple regression analysis on the effect of TGF-ß1, EGF, FGF and estrogen to proliferation revealed that only TGF-ß1 had negative feedback. This indicated that TGF-ß1 decreased, so that the proliferation increased. Estrogen had positive impact in proliferation, indicating that increased estrogen would also increase proliferation. In conclusion, estrogen increased the proliferation of the prostate cell and EGF significantly and decreased the expression of TGF-ß1 significantly. This leads to inhibition of proliferation, and finally may cause the occurrence of BPH.

Keyword: prostate, estrogen, TGF-ÄŸ1, EGF, FGF, BPH,

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