Benign Prostatic Hyperplasia vs. Prostate Cancer

Scene 2: *Benign Prostatic Hyperplasia vs Prostatic Cancer*

Throughout life, the male sex hormone, testosterone, influences prostate growth and development.

Prostatic stromal cells express an enzyme that converts testosterone to dihydrotestosterone.

Dihydrotestosterone binds to stromal cells, releasing signaling factors that stimulate prostatic cell division and growth.

As men age, dihydrotestosterone can accumulate among prostate cells.

In addition, circulating testosterone levels fall, and estrogen levels rise.

These hormonal changes may lead to excessive prostatic cell growth characteristic of benign prostatic hyperplasia and prostate cancer.

In benign prostatic hyperplasia, the glandular epithelium and stroma proliferate in the transition zone, proliferate and form nodules that expand the tissue, and prevent normal urination.

In contrast, prostate cancer commonly occurs in the peripheral zone, in which the glandular epithelial cells mutate and divide in an uncontrolled manner. Many new abnormal glands and one or more malignant tumors develop.

The large tumors may compress the urethra and disrupt the urine stream.

In a process called metastasis, the tumor cells spread to tissues both near and distant from the prostate.