Role of Finesteride on Benign Prostatic Hyperplasia

Androgenic stimulation is essential for benign hyperplasia of prostate (BPH). More than 95% of androgen is of testicular origin and rest 5% comes from adrenal gland. Androgenic stimulation is also essential for development of prostate and other male sex organs. Man castrated before puberty never develop BPH and castration after the disease shrinks the size of the prostrate. Testosterone do not act on prostate directly, rather it acts as prohormone. Testosterone is converted to dihydrotestosterone (DHT) in the prostate by the enzyme 5-α reductase to DHT which is about ten times more potent than testosterone[1]. There is also five fold higher concentration of DHT than testosterone in prostatic diseases. DHT binds with specific androgen receptors in epithelial stromal cells and regulates a variety of process like growth, differentiation, function of prostate, and it also stimulates BPH.

Finesteride is an anti-androgen that competitively blocks 5-α reductase, the enzyme which converts testosterone to DHT. It is an one way process and is irreversible. There are two isoenzymes of DHT. Type-I is present mainly in skin, scalp, liver and to a lesser extent in prostate. It is believed to be involved in hair growth. Type-II isoenzyme is mainly present in prostate gland and other accessory male genital organs. Type-II enzyme appears in the basal cells of the epithelium and stromal cells but is absent in secretory epithelium. Finesteride, when used for a longer time, causes 20% shrinkage of the size of the prostate gland and reduces prostate specific antigen (PSA) by 30%. Its peak action in reducing the size occurs after six months and urinary flow rate improves in two months time.

Finesteride improves urinary flow rate, reduces prostatic obstruction and symptoms, and relieves episodes of acute retention of urine. It also improves recurrent gross haematuria due to BPH. Finesteride is also helpful in the treatment of androgenic alopecia. Long time use of finesteride also reduces the incidence of carcinoma of prostate by 25%[2]. Principal side effects of finesteride includes loss of libido, ejaculatory dysfunction and impotence in less than 5% cases.

Microscopic changes that occur following 12 months treatment with finesteride causes shrinkage of the size of stromal cells and increases apoptosis. The article published in this issue regarding the effects of finesteride on rat prostate has substantiated the already established facts[3].

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