Haider S. Jaffat et al /J. Pharm. Sci. & Res. Vol. 10(1), 2018, 132-133

ISSN:0975-1459
Journal of Pharmaceutical Sciences and Research
www.jpsr.pharmainfo.in

Determination of Total Antioxidant Capacity, LH, FSH and Testosterone in Serum of Male Albino Rats which orally given by Finasteride (Prostacare)

Haider S. Jaffat * and Fahad N. Obaid
Biology Department, Faculty of Sciences, University of Kufa, Iraq.

Abstract:

This study was led to explore the impact of finasteride (Prostacare) on testicular capacity in *Rattus Norvegicus*. Forty five matured male rats with body weight of (200-300g) and (8) to (10) weeks of age were divided into three groups (15 rats per group). The first group was orally given with distilled water as a control and the others (second and third) were orally given with two concentrations of Finasteride (0.014mg. and 0.028mg) daily for a period of (eight weeks). After the finish of the experiment ,rats was scarified to obtain blood samples (2.0– 5.0 ml) immediately after sacrificing by heart rupture and positioned into Eppendrof tubes and allowable to clot. The results show decrease in total antioxidant capacity in blood serum with two concentrations ,but not significant ($p<0.05$) when comparing with the control group.

Key words: Sexual dysfunction , Finasteride , Testosterone , TAC.

INTRODUCTION:

Finasteride is an inhibitor of human five-α reductase causing in the reduction of DHT creation [1]. Finasteride is an inhibitor of five-α reductase enzyme type-two, preventing the transformation of testosterone to dihydrotestosterone (DHT), Finasteride used as an oral drug for the usage of benign prostatic hyperplasia at the 5mg /day dose and is also used for the treatment of androgenic alopecia, or hair loss [2].

The chemical name of Finasteride is N-tert-Butyl-3-oxo-4-aza-5-androst-1-ene-17-carboxamide. The empirical formula of Finasteride is C23H36N2O2 and its molecular weight is 372.55, Finasteride is a white crystalline powder with a melting point near 250°C. It is freely soluble in chloroform and in lower alcohol solvents but is practically insoluble in water [3]. Any deviations in the Testosterone/DHT percentage in male offspring born from females fertilized by Finasteride-treated male rats can product in impairment of testicular physiology [4].

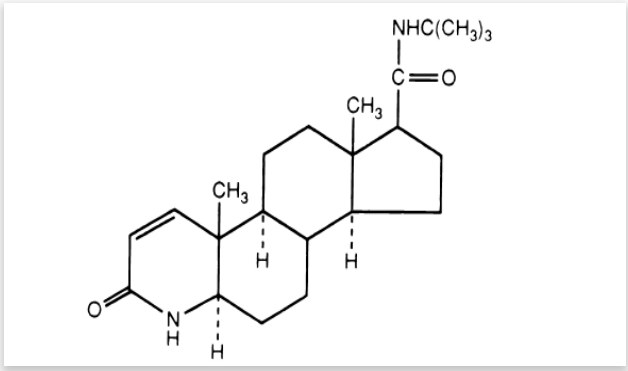

Figure 1 Chemical structure of Finasteride.

MATERIAL AND METHODS:

Chemicals and reagents:

Finasteride (Prostacare-5mg) which daily dose for Human. Matured male albino rats were used for this experimental study. The treated groups with prostacare with two doses (0.014mg and 0.028mg) were administered orally to male albino rats using separate sterilized oral dosing needle for a period of eight weeks, were therapeutic doses calculated for the weight of the rats. The control group received the same volume (distilled water) alone. TAC Kit ,FSH Kit, LH Kit and Testosterone Kit are used in this study which company Suppliers by Monobind company (USA).

Instrumentation:

Total antioxidant capacity tests were measured by ferric reducing–antioxidant power (FRAP) method, which is exact widely used today [5]. The FRAP assay is constructed on the reduction of ferric ions to ferrous ions by the effect of the reducing power of the plasima (or a samplie) constituents measuried by spectrophotometer at 593nm [6].

The assay, rat Follicle Stimulating Hormone (FSH) hormone ELISA and rat Luteinizing Hormone (LH) hormone ELISA ,Kits were conducted according to the manufacturing company (Monobind company) that depended on the technique of the quantitative sandwich enzyme immunoassay.

Serum testosterone was measured by utilizing Mini VIDAS (Biomeieusvitek ,USA). The examine standard consolidates a catalyst immunoassay rivalry technique with a last fluorescent location (Enzyme Linked Fluorescent Assay). The conjugate enizyme catalyzes the hydrolysis of this substrate into a fluorescent item (4-Methyl-umbelliferone), fluorescence was then measured at 450 nm. The power of the fluorescence is contrarily corresponding to the centralization of testosterone introduce in the specimen. Toward the finish of the examine, comes about were consequently figured by the instrument in connection to the alignment bend put away in memory and after that printed out.

Experimental Animals:

Forty five mature, healthy, fertile and adults albino male rats (*Rattus Norvegicus*) aged (8-10) weeks were obtained from the animal house of College of Veterinary/ Al-Qadisiyah University . The weight range was (200-300) gm. The animals were housed in plastic caged .The caged were embedded with wooden shelves, under natural (12hr) light and (12hr)dark, The animals were caged at lab temperature of (23 – 25°C), and the animals were feed *ad libitum* . The animals were reproduced to obtained a propitiate numbers of animals. They were divided into three groups (15 animals/group).

Laboratory animals:

Animals weight has been recorded before and after the dosage by using electrical balance. Animals were Sacrifice by cervical dislocation, Immediately after Sacrificed the abdominal cavity was opened in overturned (T) shape to obtain blood samples (2.0–5.0 ml) immediately after sacrificing by heart rupture and positioned into eppendrof tubes and allowable to clot and Serum was separated by centrifuge for ten minutes at 3000 rpm. The isolated serum was kept at -20°C until the measurement of TAC , LH ,FSH and testosterone hormone.

Table 1: The effect of different concentrations of Finasteride (Prostacare) 0.014mg and 0.028mg on Testosterone, LH ,FSH and TAC compared with control group.

Treatments	Parameter			
	Testosterone Levels(mlU/ml) (Mean ± SEM)	LH levels(mlU/ml) (Mean ± SEM)	FSH levels(mlU/ml) (Mean ± SEM)	Total antioxidant Capacity Levels(µm/L) (Mean ± SEM)
Control	4.386 ± 0.128	4.114 ± 0.055	3.200 ± 0.053	669.978 ± 1.261
Prostacare (0.014mg)	3.171 ± 0.068 [a]	3.243 ± 0.053 [a]	2.400 ± 0.082 [a]	645.535 ± 1.485
Prostacare (0.028mg)	2.100 ± 0.044 [ab]	1.457 ± 0.065 [ab]	1.543 ±0.065 [ab]	615.495 ± 1.307
L.S.D.	0.056	0.058	0.071	N.S

a = representing a significant (P<0.05) differences in comparison to control. b = representing a significant (P<0.05) differences in comparison to between two treatments (0.014mg and 0.028mg). N.S= NO Significant

Statistical analysis:

The results were expressed as (Mean ± Standard Error). One -way ANOVA was used for the comparison between control and other groups in the measured parameters. followed by least significant difference (L.S.D) analyses at 0.05% probability of levels. All statistical analysis was performed using Excel program (2010) and Magastat program .The test of significance was placed at ($p<0.05$).

RESULTS:

The results demonstrate there were reduction, but not a significant ($p<0.05$) in the Total antioxidant level in the serum of the groups administered with (0.014mg.) and (0.028mg) of Prostacare. Results demonstrate there were a significant ($p<0.05$) reduction in the Testosterone levels in the serum of the group administer by (0.014mg) and (0.028mg) of Prostacare. There were too a significant decrease ($p<0.05$) in Testosterone levels between treatments. **Table (1)**

The results, also shown a significant decrease ($P <0.05$) in the concentration of LH and FSH in the first and second treatment groups (0.014mg) and (0.028mg) of Prostacare compared to the control group.**Table (1)**.

.

DISCUSSION:

Reduced total equivalent antioxidants capacitiy levels were found in patients affected by BPH compared with controls [7].The total antioxidant capacity was significantly higher in the control group than in any other infertile groups [8].

In normal state, Testosterone is controlled by a negative feedback mechanism, the diminish of testosterone hormone cause increase in the level of LH and FSH. The hypothalamic secretes gonadotropin-discharging hormone (GnRH) that follows up on the front pituitary to deliver follicle-animating hormone (FSH) and luteinizing hormone (LH) [9]. In this study Finasteride cause decrease LH may because impact to the interstitial Leydig cells of the testicles, also decrease FSH may be through spermatogenesis, Finasteride which effect on Sertoli cell work.

The level of testosterone decreases through either the effect on steroidogenesis enzymes in testes , or its inactivation properties on adrenergic systems involved in steroidogenesis .This effect can be explained by the anti-androgenic action as well as the stimulation of steroidal anti-androgen to the negative feedback inhibition of the hypothalamus which resulted in lowering the concentration of plasma testosterone [10].

These decrease in testosterone level can also be explained by necrosis induced in the seminiferous tubule ,the significant decrease in interstitial cells number lead to decreased testosterone secretion from interstitial cells [11].

CONCLUSION:

Our conclusion of finding results shown a significant ($p<0.05$) decreases in the levels of testosterone, Luteinizing hormone(LH) and Follicle stimulating hormone(FSH) in blood serum with two concentrations of Finasteride comparing with the control group .

REFERENCES:

1. Sheikh, S.; Ahmad A. ; Ali, S.M ; Ahmad M.U.; Paithankar, M. ; Saptarshi, D.; Kale, P.; Maheshwari, K. ; Hemant V. B.; Pathak H.L; Mohammad, M. and Ahmad, I..(2015). A New Topical Formulation of Minoxidil and Finasteride Improves Hair Growth in Men with Androgenetic Alopecia , J Clin Exp Dermatol Res, 6:1.Michael S. Irwig. (2013). Decreased Alcohol Consumption Among Former Male Users of Finasteride with Persistent Sexual Side Effects : A Preliminary Report, 1–4.
2. Wu, C.; Forbes, E. and Jarvi, K. A. (2017). RESIDENTS ' ROOM Clomiphene citrate rescue of spermatogenesis in men with infertility while remaining on finasteride : A case report, *11*(April), 122–123.
3. Merck, S. and Dohme, C. (2011). PROPECIA ® (finasteride) Tablets FULL PRESCRIBING INFORMATION INDICATIONS AND USAGE PROPECIA ® is indicated for the treatment of male pattern hair loss (androgenetic) in MEN ONLY . Efficacy in bitemporal recession..
4. Kolasa-Wołosiuk, A., Misiakiewicz-Has, K., Baranowska-Bosiacka, I., Gutowska, I., Tarnowski, M., Tkacz, M., & Wiszniewska, B. (2016). Connexin 43 expression in the testes during postnatal development of finasteride-treated male rat offspring. *Archives of Medical Science*, *2*, 1–9.
5. Szeto, Y.T.; Tomlinson, B. and Benzie, I.F.F. (2002). Total Anti-oxidant and ascorbic acid content of fresh fruits and vegetables: Implications for dietary plan-ning and food preservation. Brit J Nutr. 87: 55-9.
6. Carlos, K. and Bucalen, F.(2007). Total Antioxidant Capacity: a biomarker in biomedical and nutritional studies). End. Res: R. Cardeal Arcoverde, 1663 – apto 41, 05407-002 ,Pinheiros, Sao Paulo (SP), Brazil.
7. Minciullo, P. L.; Inferrera, A.; Navarra, M.; Calapai, G.; Magno, C. and Gangemi, S. (2015). Oxidative stress in benign prostatic hyperplasia: A systematic review. *Urologia Internationalis*, *94*(3), 249–254.
8. Sharma, R. K.; Pasqualotto, F. F.; Nelson, D. R.; Thomas, a J. and Agarwal, A. (1999). The reactive oxygen species-total antioxidant capacity score is a new measure of oxidative stress to predict male infertility. *Human Reproduction (Oxford, England)*, *14*(11), 2801–2807.
9. Dandona, P. and Rosenberg, M. T. (2010). A practical guide to male hypogonadism in the primary care setting. *International Journal of Clinical Practice*, *64*(6), 682–696.
10. Mocktary, M.; Shariati, M. and Amiri, J. (2007). Effect of Tamsulosin on serum testosterone and gonadotropins concentration in adult male rats. *Journal of Rafsenjan university of medical sciences*., PP:6(1):1-6.
11. Hibi, H.; Yamamoto, M. and Miyake, K . (1995). Effects of Alpha-Blocker on sperm concentration, motility, intra luminal pressure and fluid movement in the rat cauda epididymis ,*J. Urol* .154(2) :606-610.