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Measuring the Level of the Happiness Hormone Serotonin, Dehydroepiandrosterone and Some Biochemical Variables in Patients with Enlarged Prostate

Hiba H Rasheed[1]*, Safa A Abdulqader[1], Shaimaa M Abdolrazzak[2] and Firas S Alguri[3]

[1]College of Science, University of Tikrit, Iraq
[2]Ministry of Education, Education Department, Iraq
[3]College of Medicine, University of Tikrit, Iraq

***Corresponding author:** Hiba H Rasheed, College of Science, University of Tikrit, Iraq, Email: Hibarasheed@tu.edu.iq

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Abstract

An enlarged prostate indicates that the gland has become bigger. As men age, virtually all of them develop prostate enlargement, which is commonly referred to as benign prostatic hyperplasia (BPH). The study found that greater levels of the hormone DHEA in prostate serum may predict the existence of individuals with benign prostatic hyperplasia or prostatitis as compared to healthy people. 5-hydroxytreptamine, which is related with prostatitis and enlargement, was also investigated, and we found that patients had lower levels of 5-hydroxytreptamine than controls. Prostatitis and hypertrophy were far More common as compared to a control groups ($P<0.05$). Their impact on a variety of markers, including albumin, creatinine, HDL triglyceride and demographic parameters, blood pressure, diabetes, age, smoking, the effectiveness of therapy, the types of therapies employed, body mass, the environment.

Keywords: DHEA, Prostate Enlargement; Prostatitis; Benign Prostatic Hypertrophy(BPH)

Introduction

At birth, the prostate is small and remains so until early puberty when it expands in size during an androgen-dependent pubescent development phase [1], reaching (20±6g) [2,3]. Any further is pathological and described clinically as benign prostatic enlargement (BPE), which occurs in roughly 50% of men by age 50 and 90% of men over the age of 80 [4,5].

Lower urinary tract symptoms (LUTS) is the whole mark of BPH, with symptoms ranging from nocturnal, incomplete emptying, urine hesitancy, weak stream, frequency, and urgency to severe urinary retention [6,7]. The conventional cause of BPH is hormones and genetic susceptibility, while modifiable risk factors include obesity, high fasting plasma glucose, cardiovascular disease, diabetes, dyslipidemia, and the metabolic syndrome [8]. Prostatitis pelvic pain syndrome is classified, and it is notoriously difficult to treat, with a recurrence incidence of up to 50% [9,10]. Although serotonin (5-hydroxytryptamine or 5-HT) was discovered 60 years ago [11,12]. Neuroendocrine prostatic cells produce a variety of neuroendocrine substances, the most prevalent of which is 5-HT [13]. Notably, neuroendocrine prostatic cells are mostly found in the transition zone of the normal human prostate [14], which is where BPH develops [15]. However, when comparing BPH tissue to a normal transition zone (without BPH), the number of neuroendocrine cells is

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significantly reduced [16-18]. Additionally, 5-HT was shown to be considerably reduced in BPH tissue [19]. Furthermore, a recent investigation in a large cohort of Scandinavian males found that LUTS is related with benign prostate enlargement and lower plasmatic 5-HT concentrations [20]. These findings point to a potential relationship between prostatic 5-HT depletion and BPH pathogenesis [21]. DHEA and DHEAS have also been identified as significant neuroactive hormones [22-24]. Prostate cells use enzymes 17β-hydroxysteroid dehydrogenase (17β-HSD), 3β-HSD, and 5α-reductase to regulate the quantity of active sex hormones inside the cell [25]. It is believed that the adrenal steroid DHEA is a major source of androgens, which, when digested by prostate cells, provide up to one-sixth of the DHT present in the prostate [26].

Materials and Methods

From February 2022 to October 2023, sera were collected from 50 individuals with benign prostatic hyperplasia and 50 individuals having prostatitis. as well as 45 healthy controls. From a hospital. The patients' ages varied from 32 to 85, with an average of 73.09±8.38 years, and a medical history spanning 1 to 11 years. Five individuals experienced acute urine retention. The sample was transported to the laboratory to be separated into serum and plasma using a separator. It was then placed in a deep freeze to investigate the impact of the parameters on the samples. Determination of human hormone serotonin activity: Serum hormone serotonin activity has been determined by using Kit assayed according to the manufactured Procedure (Bioassay technology Laboratory, Cat.NO EA0028Hu, China). Determination of Human dehydroepiandrosterone ELISA Kit: Serum dehydroepiandrosterone. The activity was measured using a kit (Fine Test Laboratory, Cat. No: EH4005, China), following the manufacturer's instructions. Statistical analysis: The biochemical data were statistically evaluated using the statistical software tool SPSS17.0. The mean standard deviation (SD) was estimated using ANOVA, Statistical significance was determined when the P value was equal to or less than $p < 0.05$.

Results

This study demonstrated that the concentration dehydroepiandrosterone in prostate enlargement and prostatitis significantly increased in compared with control. The results showed that the concentration dehydroepiandrosterone increase creatinine and albumin significantly in prostate enlargement and prostatitis patients compared to the control. They also showed that the decreased concentration dehydroepiandrosterone also decrease good fats and increase TG compared with the control (Table 1). Moreover, the study showed that the serotonin concentration significantly decreased in prostate enlargement and prostatitis compared with control. The results showed that the decreased concentration of serotonin also associated with decreased creatinine, and HDL while increases TG in prostate enlargement and prostatitis compared with the control (Table 1).

Parameters	Prostate Enlargement	Prostatitis	Control	P value
Dehydroepiandrosterone (ng/ml)	236.2±50*	313±54.1*^	160.44±52.46	0.0005
Creatinine (U/L)	204±44.71*	206±45.31*	190±38.31	0.0007
Albumin (g/L)	228±51.72*^	219±49.21*	187±35.24	0.0004
HDL (mg/L)	174±31.2	183±30.12	203±38.76*	0.00051
Triglyceride(mg/L)	230±50.73*	236±53.22*	214±34.78	0.00043
parameters	Prostate enlargement	Prostatitis	Control	P value
Hormone serotonin (µg/dL)	27.23±5.84	25.33±5.02	44.72±6.32*	0.0009
Creatinine (U/L)	25.61±5.89	23.22±5.1	43.31±5.81*	0.0006
Albumin (g/L)	24.62±5	20.31±4.22	31.82±6.71*	0.0003
HDL(mg/L)	19.71±1.82	20.88±2.12	48.21±3.52*	0.00081
Triglyceride(mg/L)	26.21±4.76	24.56±3.88	29.73±2.17*	0.0005
Data expressed as mean ±SD, *^ Significant changes ($p<0.05$) were seen as compared to the control or prostatitis groups, ^ as compared to control group.				

Table 1: Serum concentrations of dehydroepiandrosterone, serotonin, and parameters in the examined groups.

At age (30-45), the dehydroepiandrosterone increased in prostatitis patients compared with prostate enlargement. Conversely, dehydroepiandrosterone increased in prostate enlargement compared to prostatitis patients at age (46-85). In normal and overweight, dehydroepiandrosterone increased in prostate enlargement compared to prostatitis patients. Conversely, dehydroepiandrosterone increased in prostatitis patients compared with prostate enlargement in case of obesity group. Smoking has no impact on the level of dehydroepiandrosterone despite of increased levels in prostate enlargement compared to prostatitis in smoking-free. Compiling diseases significantly increases dehydroepiandrosterone In patients prostate enlargement with diseases increase a significant compared with diseases patients. While a significant decrease in patients prostatitis with diseases compared without diseases. In prostate enlargement, increase -significant in rural areas compared in locations rural compared with patients in urban and increase in prostatitis patients significant urban compared with prostatitis patients rural. In prostate enlargement and prostatitis, patients without genetic link no a significant. compared without genetic factor increase significant. Hypertension has non-significantly changed the level of dehydroepiandrosterone in either group of prostate enlargement or prostatitis, compered no hypertension decreased a significant prostatitis patients. Hyperglycemia has significantly elevated serum of dehydroepiandrosterone levels in prostate enlargement increase a significant compared with no hyperglycemia while in patients prostatitis hyperglycemia decrease a significant compered no hyperglycemia (Table 2, Figure 1).

Prostate enlargement	Prostatitis	Demographic Parameters	
383.9 ±68.3	440.1 ±83.3	30-45	Age (years)
277.4 ±94.7	185.2 ±42.5	46-85	
261.9 ±24.1 a	257.2 ±77.6 b	Normal	BMI
282.9 ±42.7 a	237.8 ±70.5 b	Over Weight	
237.8 ±65.1 a	265.5 ±52.0 b	Obese	
255.0 ±57.7	254.2 ±53.2	Positive	Smoking status
387.2 ±43.1	250.0 ±53.0	Negative	
282.7 ±51.8	300.4 ±50.0	Positive	Diseases status
259.1± 65.4	304.6 ±77.5	Negative	
318.9 ±81.2	260.7±70.4	Rural	Geography
321.8 ±76.7	232.4 ±86.7	Urban	
207.7 ±58.1	209.0 ±57.3	Positive	Genetic status
302.0 ±63.1	405.2 ±64.7	Negative	
204.2 ± 63.0	254.7 ± 68.9	Positive	Hypertension
198.7±64.65	185.9±43.1	Negative	
261.6±83.0	105.8±61.96	Positive	Hyperglycemia
241.3±47.90	389.5±77.8	Negative	
244.3 ± 78.3	--------	Urimax capsule	Therapy Current
253.3 ±79.9	----------	Xradal tablet	
----------	289.4 ± 87.5	Prostacalm capsule	
----------	205±79.23	Prostanil tablets	

Data expressed as mean±SD, different letter indicate significant differences at $p<0.05$ within the same group, *indicate significant differences at $p<0.05$ within between the two group,. prostanil=Finasteride, Prostcalm=a combination of natural plant extracts with organic and mineral antioxidants, Xradal =alfuzosin, Urimax= tamsulosin

Table 2: Serum concentration of dehydroepiandrosterone relative to their demographic parameters in patinate groups.

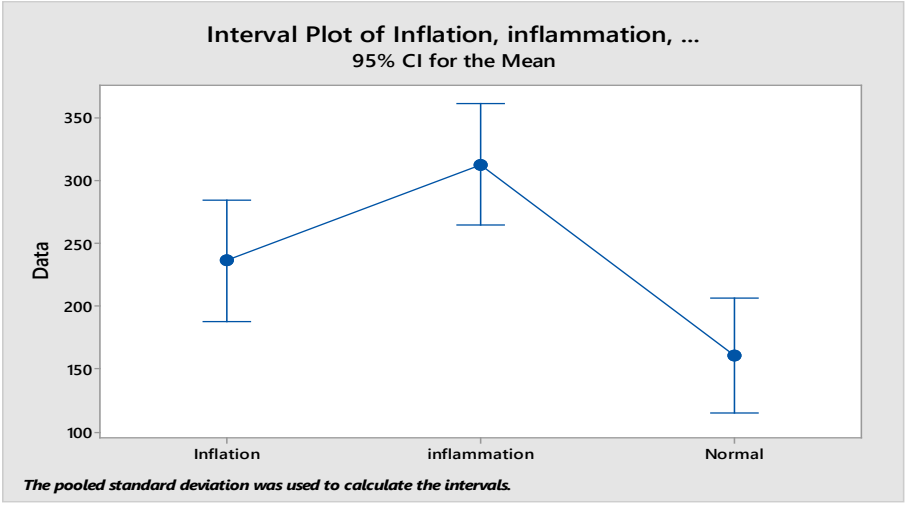

Figure 1: Concentration of dehydroepiandrosterone in patinates compered normal.

The effect of hormone serotonin in patinate prostate enlargement no a significant in age (30-45) compered in age and (46-85), while in prostatitis patinate the result showed deceased a significant in age (30-45) compered in age (46-85) .on the body mass of enlarged prostate patients. There is a significant increase in patients group a obese bodies compared to prostate enlargement who are overweight and patients normal, while prostatitis patients group b a significant increase in over wait compered the obese body, and normal body. in patients prostate enlargement with genetic factor no a significant compared patients without genetic factor, while increase significant in prostatitis patients without genetic factor compered patients without genetic factor (26.38±3.32). no significant in patients prostate enlargement smoking compared to not smoking. But increase a significant in patients prostatitis not smoking compared to patients prostatitis smoking in geographical between rural and urban we noticed increase significant in patients prostate enlargement in urban compared with patients in rural and increase significant(34.87 ±19.09)of prostatitis patients in rural compared with prostatitis patients (25.76 ±11.53) urban. patients prostate enlargement no a significant(23.23 ±2.98) compared without diseases patients (20.53±1.73) While a significant increase (24.88±4.85) in patients prostatitis with diseases compared without diseases (20.97±3.80). in patients prostate enlargement with high blood pressure no a significant(21.55 ±2.95) compered without blood pressure (23.32 ± 3.02). in patients prostatitis without high blood pressure increased a significant (44.31±7.45)compared with high blood pressure (3.13 ±26.29). In patients prostate enlargement with high blood sugar increase a significant (16.27±14.46) compered without blood sugar (27.93±5.54). in patients prostatitis with high blood sugar increase a significant (25.83±48.41) compered without blood sugar (28.00±42.90). In patients prostate enlargement who use kind drag urimxe increased a significant(29.99±5.72) compered who use kind drag xradal (2.84±26.36),While in Table 3 in patients prostatitis who use kind drag prostalin showed increase in a significant (30.67 ±22.59)compered patients who use prost calm (3.53±23.93) (Figure 2).

Prostate Enlargement	Prostatitis	Demographic Parameters	
23.14 ±3.08	23.47±4.7	30 – 45	Age (years)
22.67 ±3.38	30.62±5	46-85	
25.59 ±4.27 a	16.74±2.77b	Normal	BMI
04.3. ±26.35	24.2± 25.75	Over Weight	
42.6±7.30 a	18±3.33b	Obese	
30.8±4.37	18.73±3.02b	Positive	Smoking status
21.99±4.3	27.53±6.86b	Negative	

23.23±2.98	24.88±4.85	Positive	Diseases status
20.53±1.73	20.97±3.80	Negative	
28.55±13.08	34.87±19.09	Rural	Geography
27.15±17.18	25.76±11.53	Urban	
29.58±4.92	17.75±4.77	Positive	Genetic status
19.99±4.59	3.32±26.38	Negative	
21.55±2.95	26.29±3.13	Positive	Hypertension
23.32 ± 3.02	44.31±7.45	Negative	
16.27±14.46	25.83±48.41	Positive	Hyperglycemia
27. 93±5.54	28±42.90	Negative	
29.99±5.72	------	Urimax capsule	Therapy Current
26.36±2.84	-------	Xradal tablet	
--------	23.93±3.53	Prostacalm capsule	
--------	30.67±22.59	Prostanil tablets	
Data expressed as mean±SD, different letter indicate significant differences at p<0.05 within the same group, * There are substantial differences (p<0.05) between the two groups.			
prostanil= Finasteride, Prostcalm=a combination of natural plant extracts with organic and mineral antioxidants, Xradal =alfuzosin, Urimax= tamsulosin			

Table 3: serum concentration of hormone serotonin relative to their demographic parameters in patinate groups.

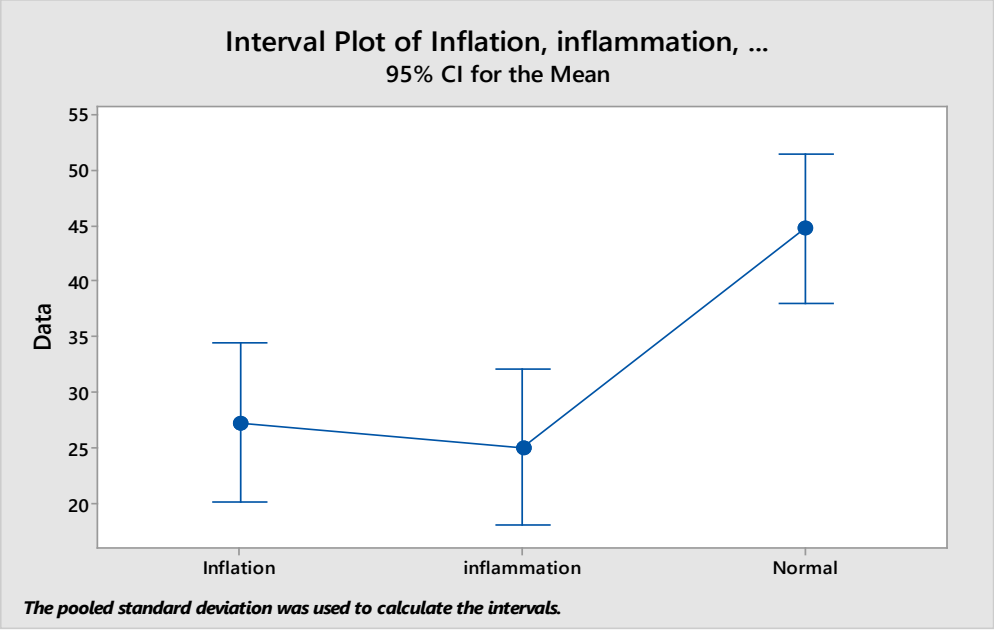

Figure 2: Concentration of hormone serotonin in patinate compered normal.

Discussion

Neuroendocrine prostatic cells produce a range of neuroendocrine chemicals, with 5-HT being the most common. Some neuroendocrine cells have a unique morphology, with dendritic processes extending into the lumen and projections surrounding the epithelial-stroma interface, which supports the idea that neuroendocrine products, such as 5-HT, may impact prostate development [27]. Particularly, neuroendocrine prostatic cells are mostly

located in the transition zone of the normal human prostate [28], where BPH occurs [29]. However, when comparing BPH tissue to normal transition zone (without BPH), the number of neuroendocrine. While compare BPH tissue to the normal transition zone (without BPH), the number of neuroendocrine cells is much lower [30,31]. Serum creatinine and albumin are muscle metabolites that are directly related to the body's total muscle mass. Serum creatinine concentration is an effective biomarker for assessing glomerular filtration rate (GFR), which is important for clinical evaluation and therapy [32]. The purpose of this study was to look at the impact of creatinine in serum 5-HT and DHEA levels in predictive risk stratification in prostate patients in order to give guidance for clinical decision-making. Lipids in the blood and prostate incidence and prognosis are being investigated at the genetics and medical stages. Andreassen, et al. [33]. The results revealed that the hormone serotonin no a significant in age in the age group of 46-85 and younger age group in patients with prostate enlargement, this implies that the hormone does not grow with age, but in patients prostatitis increased hormone in age 46-85 compared to the younger age group. We discovered that patients with prostatitis and enlargement have higher levels of serotonin than healthy persons, which is consistent with the findings of researchers Emanuel Carvalho-Dias and Alice Miranda [34]. people with prostatitis and prostate enlargement had higher levels of the hormone serotonin compared to normal and obese people. Hormone concentrations were not significantly different in smoking individuals with prostate enlargement compared to non-smokers, indicating that prostate enlargement is associated with smoking, as previously reported. Patients with prostatitis are more likely to be nonsmokers than smokers. Some researchers show a modest negative relationship cigarette smoking and BPH, either diagnosed by symptoms or clinical evaluation [35-37]. Some studies even suggest that smoking has a preventive effect on BPH [38,39]. Many believe that cigarette smoking increases the complications of BPH. Once more, many urologists believe that the effect of smoking tobacco on BPH is proportional with the quantity smoked [40,41].

Condition status results revealed that the hormone serotonin rose in people with prostatitis and prostate enlargement compared to those without the condition. The geographical location plays a part in the increase in the concentration of the hormone serotonin in prostate growth .Patients who reside in cities have higher hormone concentrations than those who live in rural areas, while the converse is true for prostate patients. The genetic factor has an influence on the hormone serotonin prostate enlargement, no a significant difference between who do not have a hereditary family history and who have a family history, but in patients prostatitis, increased in the family hereditary factor compared to do not have a hereditary family history. This suggests that the hereditary component does not impact hormone concentration in people with an enlarged prostate. hypertension in patients with prostate enlargement, increased serotonin concentrations without blood pressure compared to those with blood pressure, but in patients prostatitis without blood pressure increased hormone serotonin, of the hormone in patients who suffer from blood pressure compared to those who do not. Individual met components such as type 2 diabetes and hypertension were investigated [42]. Hyperglycemia in patients with prostate enlargement and patients with prostatitis increases hormone in comparison to those without hyperglycemia. Diabetes is commonly associated with low PSA levels; earlier studies imply a relationship between metS and BPH [43]. Hyperglycemia is linked to reduced parasympathetic activation through neuronal death [44]. A disparity between sympathetic and parasympathetic activity can result in bladder neck obstruction and reduced urine power. The Rho kinase pathway is essential for prostate contraction [45]. Androgens are crucial for the proper prostate's formation, growth, and function [46].

The adrenal cortex secretes Dehydroepiandrosterone (DHEA) and its sulfate counterpart, DHEA sulfate (DHEAS), are among the most common steroid hormones among humans [47,48]. Previous studies have shown that DHEA may be associated with the development of cancer of the prostate [49,50]. However, the usefulness of blood DHEA levels necessary for prostate cancer screening remains uncertain. The results also indicated that the hormone serotonin increases in the age group of 46 to 85 in individuals with prostate enlargement compared to The smaller age range. In contrast to inflammatory patients, the hormone increases with age. The hormone rises in a smaller age group than older people.

The concentration of the hormone increases in obese and overweight patients with prostate enlargement, and decreases in those with normal weight. A BMI greater than 35 kg/m^2 was associated with a 3.5-fold higher risk of acquiring an enlarged prostate volume (>40 ml) [50]. Individuals with a BMI < 25 kg/m^2 were 1.2 times more likely to have LUTS [51]. Hormone concentrations in smokers with prostate enlargement were lower than in nonsmokers, showing that smoking caused prostate enlargement, as previously documented. In the presence of other diseases associated with prostate enlargement, There has been no significant change in the levels of the hormone serotonin among smokes, and nonsmokers; however, prostatitis patients had a higher concentration of the hormone than patients without other diseases. Individuals with a BMI < 25 kg/m^2 were 1.2 times more likely to develop LUTS [52]. Hormone concentrations in smokers with prostate enlargement were lower than in nonsmokers, indicating that smoking causes prostate

enlargement, as previously reported. In the presence of other disorders linked with prostate enlargement, there was no significant difference in the concentration of the hormone serotonin between smokers and nonsmokers; however, individuals with prostatitis had a greater concentration of the hormone than those without the condition. Hypertension in individuals with prostate enlargement and prostatitis raises concentrations without blood pressure compared to those with blood pressure. We found that patients with prostatitis had a greater concentration. Hyperglycemia in patients with prostate enlargement and patients with prostatitis increases hormone in comparison to those without hyperglycemia. Individuals with excess visceral fat are more likely to develop insulin resistance, which may be an independent risk factor for BPH [53,54]. As a result, obese, dyslipidemic, and older men are at risk for MetS, which has components that are good indicators of prostate growth [55,56]. Other comorbidities linked to abdominal obesity include cardiovascular disease, obstructive sleep apnea, and nonalcoholic fatty liver disease [44-58]. Marberger, et al. discovered that high amounts of 5α-reductase and DHT in the prostate can cause the development and progression of BPH, even with low circulating T levels [59]. Dutasteride was discovered to be useful for BPH patients at all blood testosterone levels [57,58]. On the other hand, one-year study of T treatment in hypogonadal males revealed no significant rise in PV levels [59,60].

Conclusion

These findings link hormone serotonin and dehydroepiandrosterone to human prostate pathology, and this model provides a diverse platform for investigating the molecular underpinnings of inflammation-related prostate disorders linked with episodic or chronic hormone serotonin and dehydroepiandrosterone levels. In BPH, inflammation may be taken into consideration in the best therapy selection because if discovered, patients should have a symptomatic favorable improvement by adding anti-inflammatory drugs to routine treatments., We detected a rise in the hormone serotonin, in patients with prostatitis and enlargement compared to the control, whereas we observed a reduction in dehydroepiandrosterone. The purpose The purpose of this research was to determine the role of dehydroepiandrosterone in the control of benign prostatic growth, as well as to look into pharmacologic modulation of the prostatic serotoninergic system as a possible pharmaceutical target for BPH.

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