Transrectal Ultrasound (TRUS) Findings of the Prostate Gland in Late Onset Hypogonadism with Testosterone Supplementation in Correlation with Clinical Outcome

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Objective: *To determine the TRUS findings of the prostate and correlation of ultrasound findings with clinical outcomes in late-onset hypogonadal (LOH) men with testosterone supplementation.*
Material and Method: *Between January 2007 and September 2010, TRUS findings and clinical outcomes of 16 from 226 subjects were studied. The demographic data, ultrasound parameters as prostate volume and vascularity, and clinical parameters were evaluated. Correlation between ultrasound and clinical parameters were analyzed using Pearson correlation analysis.*
Results: *During mean time follow-up of 6.48 months, the volume of the central gland (CG) significantly increased ($p = 0.02$), the volume of the total gland (TG) increased, and the volume of the peripheral zone (PZ) slightly decreased. The vascularity of the TG, CG, and PZ were significantly increased. The periurethral region vascularity was not significantly increased ($p = 0.06$), whereas total serum testosterone, prostate specific antigen (PSA), and PSA density were increased. The International Prostate Symptom Score (IPSS) was significantly decreased ($p < 0.001$). There was a significant correlation between increased prostate volume and increased serum PSA.*
Conclusion: *Testosterone supplementation in LOH men was found to cause an increase in TG volume during the first six months. The preferentially increased CG volume and prostatic vascularity might be due to exogenous testosterone. The authors observed a significantly increased PSA with a strong correlation between serum PSA and prostate volume.*

Keywords: *Prostate, Transrectal ultrasound, Late onset hypogonadism, Testosterone supplementation*

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Testosterone supplementation therapy has been used in the treatment late-onset hypogonadism (LOH) in males, which is a clinical and biochemical syndrome associated with age-related decline in testosterone level[1-4]. Adverse outcomes of testosterone therapy have been reported, including increased risk of subclinical prostate cancer, increased growth of metastatic prostate cancer, increased prostate volume, and increased serum prostatic-specific antigen (PSA)[2,5]. However, there is low frequency of prostate cancer in association with testosterone replacement therapy, and its prevalent rate is similar to that of the general population[6].

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At present, prior to initiation of testosterone supplementation all patients should be excluded from the condition of prostate cancer by undergoing digital examination, measuring serum PSA, or ultrasound (US) of the prostate[1-3,7,8]. During treatment, the patient who undergoes testosterone supplementation should be monitored for the clinical outcome of prostate disease at three, six, and 12 months, and at least annually thereafter[2].

Transrectal ultrasound (TRUS) of the prostate is a diagnostic modality that has become useful in determining prostate lesions of BPH, prostatitis, obstructive infertility, and prostate cancer[9]. However, TRUS findings of the prostate after testosterone supplementation have not yet been well described.

The aim of the present study was to determine the findings of transrectal ultrasound of the prostate particular prostate vascularity and the correlation of

ultrasound findings with clinical outcome in late-onset hypogonadal men with testosterone supplementation.

Material and Method

Patients

A retrospective review of medical records of all men with diagnosis of late onset hypogonadism (LOH) who had been treated with long acting testosterone undecanoate between January 2007 and September 2010 revealed 226 subjects. The inclusion criteria included LOH men who had TRUS performed before testosterone supplementation and had at least one follow-up TRUS. Therefore,119 subjects who had transrectal ultrasound (TRUS) performed less than two examinations and 91 subjects who had first TRUS performed after starting testosterone supplementation were excluded. The remaining 16 subjects were left in the present study. The present study was conducted with the approval of the Institutional Review Board and a waiver of patient informed consent because it was a retrospective study.

All of the included subjects were treated with parenteral testosterone at Men's Health Clinic, Ramathibodi Hospital. Injections of testosterone undecanoate (1,000 mg) were given at 0, 6 weeks, and every 12 weeks without dose adjustment according to protocols.

Sonographic examination

Transrectal ultrasound (TRUS) examination technique

The transrectal ultrasound examination of prostate was performed by one of three radiologists using a transrectal sonographic 5-9 MHz probe and Philips IU 22 system (Bothell WA, 98041 USA).

Scanning technique

The patient lay on left lateral decubitus position. The digital rectal examination was performed before probe insertion to detect rectal abnormalities. The probe was covered with condom coated outside with sonographic gel.

The probe was inserted into the rectum. First, both seminal vesicles were evaluated in transverse plane at just above the prostate base. Then, the base of prostate gland, peripheral zone, central zone, transitional zone, and fibromuscular stroma were evaluated in the transverse plane. The abnormalities were recorded.

Continuously, the prostate gland was surveyed from right side to midline to left side, by rotating the probe to the sagittal plane. The prostate volume was determined with formula: length x height x width x 0.52. The length was measured as greatest dimension in the sagittal plane, then the height (AP) was measured as perpendicular dimension in the length, and the width was measured as greatest dimension in the transverse plane of both total and central prostate volume. The central gland volume includes the anatomical regions of central zone, transitional zone, and anterior fibromuscular stroma. The peripheral zone volume was calculated by the difference between total prostate volume and central gland volume.

Power Doppler ultrasound was performed to evaluate prostate vascularity in all patients. The optimized detection of low-velocity flow was performed by setting the repetition frequency ranging from 500 to 700 Hz and a wall filter of 42-46 Hz.

Semi-quantitative measurement of prostate vascularity

Prostate vascularity was calculated by using the ImageJ that is a public domain, Java-base image processing program developed at the National Institutes of Health (agency of the United States Department of Health and Human Services). The vascularity of the total prostate, peripheral zone, central gland (including central zone, transitional zone, and anterior fibromuscular stroma) and periurethral region were calculated by drawing region of interest (ROI). The three images of each total prostate, peripheral zone and central gland were selected. The average pixel counts of three regions (total prostate, peripheral zone and central gland) as well as the periurethral region were calculated into percentage of vascularity pixels compared to the non-vascularity background pixels (Fig. 1).

Clinical evaluation

All included patients were evaluated clinically by using total serum testosterone, serum PSA, PSA density and the International Prostate Symptom Score (IPSS). The total serum testosterone was measured by commercial chemiluminescense kit (Immulite, Siemens Healthcare Diagnostics, USA). The PSA density was calculated by PSA/total prostate volume and IPSS was obtained by using IPSS questionnaire. The IPSS scores are from 0 (no complaints) to 5 (almost always) that cumulative scores of all seven questions with maximum score of 35.

Statistical analysis

Continuous variables were summarized as mean (SD) or median (range). Categorical variables

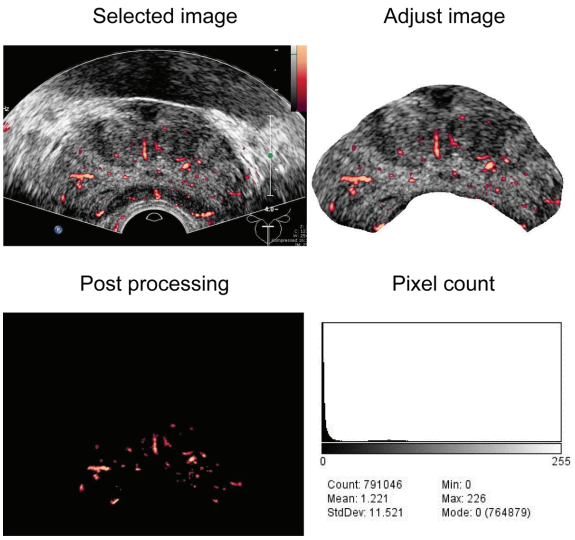

Fig. 1 Measurement of prostate vascularity by pixel count analysis using Image J post-processing software

were summarized as counts and percentage. Statistical difference of ultrasound findings and clinical outcomes in before and after treatment was analyzed using paired-samples t-test. Pearson correlation coefficient was used to determine the relationship between ultrasound findings and clinical outcomes. All p-values were two-sided and $p < 0.05$ was considered statistical significance. All statistical calculations were done using SPSS computer package version 17.0 (SPSS Inc., Chicago, IL, USA).

Results

The population consisted of 16 patients with mean age 67.11 ± 10.16 years. Fourteen patients (87.5%) had BPH, five (31.3%) had diabetes mellitus, nine (56.3%) had hypertension, eight (50.0%) had erectile dysfunction, and two (12.5%) had lower urinary tract symptoms (LUTS).

Nine patients had been treated with α1-adrenergic receptor antagonist, seven patients (77.78%) had BPH, and two patients (22.22%) had LUTS.

Seven patients had prostatic lesions at the baseline transrectal ultrasound (TRUS), in which, one lesion was pathologically proven of BPH, two were confirmed fibrosis on MRI examination, and four were unchanged after follow-up ultrasound and diagnosed as fibrosis or prostatitis and hyperplastic nodule. None had prostatic malignancy or new nodule on TRUS.

Seven patients had serum PSA more than 4 ng/ml, from which five had initial serum PSA more than 4 ng/ml before testosterone supplementation and two had serum PSA rising during testosterone supplementation. Five patients underwent prostatic transrectal ultrasound biopsy and pathological diagnosis of all of them showed benign prostatic hyperplasia. Two patients did not have prostatic biopsy performed, but they underwent MRI examination of prostate gland. One patient showed area of fibrosis or focal prostatitis at peripheral zone and one showed suspected small prostate cancer.

After testosterone supplementation, there was a significantly increased central gland volume, but not significantly increased total prostate gland volume. The peripheral zone volume was slightly decreased. The vascularity of the total prostate, central gland and peripheral zone were significantly increased. The periurethral region vascularity was not significantly increased ($p = 0.06$), whereas total serum testosterone, prostate specific antigen (PSA) and PSA density were increased. However, the international prostate symptom score was significantly decreased (Table 1).

The follow-up time was categorized into three periods as less than four months, between four and 12 months, and more than 12 months (Table 2).

The central gland volume was preferentially increased during all three periods of TRUS follow-up. All zonal prostate vascularity (total prostate, central gland, peripheral zone and periurethral region) were also increased during these three periods. The total prostate vascularity was also significantly increased. The serum testosterone was significantly increased during the testosterone supplementation, while the IPSS was decreased. The serum PSA was also increased during all periods of TRUS follow-up.

There was a significant correlation between increased prostate volume and increased serum PSA after testosterone supplementation ($p < 0.001$).

After testosterone supplementation, there had a significant decrease of IPSS in both groups of patients who was received or did not receive α1-adrenergic antagonist (Table 3).

Discussion

The present study demonstrates that the changes of prostate after testosterone supplementation could be seen by transrectal ultrasound technique. It appears that there were significantly increased central gland volume and prostate vascularity.

The study by Minnemann et al[10] has shown a significantly increased prostate volume in the first 12 months of the 25 men with parenteral testosterone

Table 1. Effect of testosterone supplementation on TRUS parameters and clinical parameters (n = 16 patients, mean time = 6.48 months)

	Mean (SD)		Change difference (95% CI)	p-value
	Baseline	After treatment		
Ultrasound parameters				
Prostate volume (cc)				
Total prostate	40.49 (24.6)	41.73 (27.7)	1.23 (-3.53 to 6.01)	0.59
Central gland	18.80 (17.2)	21.50 (18.2)	2.68 (0.46 to 4.89)	0.02
Peripheral zone	21.67 (11.0)	20.23 (10.4)	-1.44 (-6.17 to 3.30)	0.53
Prostate vascularity (%)				
Total prostate	7.35 (4.0)	15.47 (8.0)	8.13 (3.89 to 12.36)	0.001
Central gland	7.21 (5.0)	14.20 (8.8)	6.99 (2.88 to 11.09)	0.002
Peripheral zone	7.57 (4.1)	16.78 (11.2)	9.21 (3.41 to 15.02)	0.004
Periurethra region*	8.94 (12.23)	19.93 (7.84)	10.99 (-0.55 to 22.53)	0.06
Clinical parameters				
Testosterone (ng/dl)	257.06 (57.7)	574.19 (216.6)	317.13 (205.04to429.21)	<0.001
PSA (ng/ml)	2.72 (2.6)	3.14 (3.2)	0.42 (0.03 to 0.81)	0.04
PSA density	0.06 (0.04)	0.07 (0.05)	0.01 (-0.003 to 0.20)	0.15
IPSS	10.94 (7.1)	7.44 (5.7)	-3.50 (-5.16 to -1.84)	<0.001

TRUS = transrectal ultrasound; CI = confidence interval; PSA = prostate specific antigen; IPSS = international prostate symptom score

* 10 patients were missing data

undecanoate supplementation. Snyder et al[5] found a dramatically increased prostate volume during the first six months of testosterone supplementation and it was still significantly increased at 36 months of testosterone treatment. These two studies rendered support to the present results, which showed an increased prostate volume, particularly central gland volume, after testosterone supplementation.

Although the study by Emmelot-Vonk et al[11] did not show any significant change in prostate volume in the testosterone supplementation group compared with the placebo group. The present study also revealed an increased prostate volume during six months when compared to the base line.

In addition, the increased central gland volume was preferentially more than other prostatic zones during all three periods of TRUS follow-up. To the authors' knowledge, this is the first study that evaluates prostate zonal volume by dividing it into three zonal volumes (total prostate, central gland, and peripheral zone) in the late onset hypogonadal men with testosterone supplementation.

Two studies performed by Liu et al[12] and Schatzl et al[13] found two etiologic factors of BPH, including age and androgen status, from which age is the most common risk factor. Androgen has been well accepted as the second risk factor. BPH is the condition that has been described by an increase in the cellular content of the transitional zone located in the central gland[9]. It was not that most patients in the present study (87.5%) already had some degree of BPH before testosterone supplementation. The authors believe that preferentially increased of central gland volume in the present study may be due to add on BPH condition as a result of exogenous testosterone from supplementation.

There were nine patients (56.3%) in the present study who received α1-adrenergic receptor antagonist to treat obstructive symptom of BPH by relaxing smooth muscle in the prostate and the bladder neck, thus decreasing the blockage of the urine flow. The present study found that both treated and untreated groups with α1-adrenergic receptor antagonist revealed a significant decrease of IPSS representing lower urinary tract symptoms. Two studies by Permpongkosol et al[14] and Haider et al[15] also reported the effect of testosterone replacement to improve LUTS and bladder function.

Table 2. Effect of testosterone supplementation on TRUS parameters and clinical parameters categorized into three periods of follow-up

Ultrasound parameters	Follow-up time								
	Less than 4 months (n = 6)			Between 4-12 months (n = 10)			More than 12 months (n = 5)		
	Before	After	Change	Before	After	Change	Before	After	Change
Prostate volume (cc)									
Total prostate	48.8	52.9	4.02	36.8	36.6	-0.15	37.0	51.2	14.21
Central gland	26.3	29.7	3.44*	15.2	17.6	2.41	22.0	28.9	6.91
Peripheral zone	22.6	23.1	0.59	21.6	19.0	-2.56	15.0	22.3	7.30
Prostate vascularity (%)									
Total prostate	4.9	15.0	10.01*	8.6	16.6	7.96*	6.9	13.4	6.41*
Central gland	4.5	13.1	8.58	8.3	14.9	6.56*	7.8	15.8	8.09
Peripheral zone	5.6	17.4	11.80*	9.1	18.6	9.46*	5.7	8.8	3.10
Periurethra region	3.2	22.6	19.48	9.7	19.4	9.70	7.9	10.5	2.60
Clinical parameters									
Testosterone (ng/dl)	255.0	587.7	332.67*	247.9	487.1	239.20*	307.2	684.8	377.60*
PSA (ng/ml)	3.6	4.1	0.49	2.4	2.7	0.34	2.5	3.1	0.56
PSA density	0.1	0.1	0.01	0.1	0.1	0.01	0.1	0.1	-0.02
IPSS	15.2	11.0	-4.17*	10.3	6.8	-3.50*	7.0	5.8	-1.20

PSA = prostate specific antigen; IPSS = international prostate symptom score
* Statistically significant difference

Table 3. Effect of testosterone supplementation on the prostate volume and clinical parameters in patients with or without α1-adrenergic antagonist treatment

Parameters	With α1-adrenergic antagonist (n = 9)		Without α1-adrenergic antagonist (n = 7)	
	Mean change (95% CI)	p-value	Mean change (95% CI)	p-value
Prostate volume (cc)				
Total prostate	-0.71 (-7.43 to 6.02)	0.82	3.74 (-4.80 to12.28)	0.33
Central gland	3.24 (1.20 to 5.28)	0.01	1.95 (-3.34 to 7.24)	0.40
Peripheral zone	-3.95 (-10.42 to 2.52)	0.20	1.79 (-6.54 to 10.12)	0.62
Clinical parameters				
Testosterone (ng/dl)	243.22 (144.2 to 341.92)	<0.001	412.14 (167.63 to 656.65)	0.01
PSA (ng/ml)	0.43 (-0.20 to 1.06)	0.16	0.41 (-0.18 to 1.00)	0.14
PSA density	0.01 (-0.008 to 0.03)	0.19	0.003 (-0.01 to 0.02)	0.60
IPSS	-5.44 (-7.59 to -3.30)	<0.001	-1.00 (-1.93 to -0.08)	0.04

CI = confidence interval; PSA = prostate specific antigen; IPSS = international prostate symptom score

To the authors' knowledge, there has been no study so far to explain the effect of exogenous testosterone on prostate vascularity. The present study is the first study that has evaluated prostatic vascularity in the late onset hypogonadal men with testosterone supplementation in pixel count percentage. The technique can provide prostate vascularity data in a quantitative manner. The present study found a significantly increased vascularity of total prostate, central gland, and peripheral zone after testosterone supplementation. The periurethral region also showed an increased vascularity, but it was not significantly

different. This might be due to some missing data or low number of population. This result suggests that the testosterone supplementation causes increased prostatic vascularity. However, many conditions may give appearance of increased prostate vascularity such as prostatic malignancy, prostatitis, prostatic abscess, ejaculation within 24 hours, and BPH nodule in the transitional zone[16]. Additional study with a large population is needed to confirm these results.

After testosterone supplementation, serum PSA was significantly increased in the present study. However, it was not rising beyond the upper normal limit (4 ng/dl). This result was similar to the study of Gerstenbluth et al[17] that showed a significantly increased serum PSA in hypogonadal men with testosterone supplementation. It may be due to the low PSA level in hypogonadal men that responded to normal level when receiving testosterone supplementation[18]. However, serum PSA can elevate in many conditions such as prostate cancer, BPH, inflammation, prostate manipulation, biopsy, and cystoscopy[9].

In addition, the present results showed a significant correlation between increased total prostate volume, central gland volume and peripheral zone volume with increased serum PSA. The study by Roehrborn et al[19] supported the present study and they showed a strong correlation between prostate volume and serum PSA level; this relationship also depends on age.

There are several limitations in the present study. First, there is small sample size. Second, the time of TRUS follow-up varies. Third, there is incomplete information of periurethral region vascularity. Fourth, due to the operator dependent nature of ultrasound, there are variations in how TRUS is performed among radiologist that may affect prostate volume measurement and vascularity.

In conclusion, testosterone supplementation in late onset hypogonadal men was found to cause increased total prostate volume during the first six months after testosterone supplementation. The preferentially increased central gland volume may be due to exogenous testosterone and underlying BPH condition. Increased prostatic vascularity may result from exogenous testosterone supplementation and should exclude pathologic conditions. Significant increased serum PSA and a strong correlation between increased prostate volume and increased serum PSA was observed.

Potential conflicts of interest

None.

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ผลของฮอร์โมนเทสโทสเตอโรนต่อต่อมลูกหมากในผู้ป่วยที่ขาดฮอร์โมนเทสโทสเตอโรน จากการตรวจอัลตราซาวด์ของต่อมลูกหมากทางทวารหนัก เปรียบเทียบกับข้อมูลทางคลินิก

สิทธิ์ พงษ์กิจการุณ, อภินันท์ รัศมีพงศ์, สมพล เพิ่มพงศ์โกศล, มยุรีวรรณ ตั๊ะเพย, บุษณี วิบุลผลประเสริฐ

วัตถุประสงค์: *เพื่อศึกษาลักษณะการเปลี่ยนแปลงของต่อมลูกหมากด้วยการตรวจอัลตราซาวด์ของต่อมลูกหมากผ่านทางทวารหนักในผู้ป่วยที่ขาดฮอร์โมนเทสโทสเตอโรนและรับฮอร์โมนเสริม เปรียบเทียบกับอาการทางคลินิก*

วัสดุและวิธีการ: *เป็นการศึกษาย้อนหลังในผู้ป่วยที่ได้รับเทสโทสเตอโรน 16 คน ที่ได้ตรวจอัลตราซาวด์ต่อมลูกหมาก ก่อนและหลังได้รับฮอร์โมน และทำการเปรียบเทียบระหว่างขนาดของต่อมลูกหมาก ปริมาณหลอดเลือดในต่อมลูกหมาก ค่าซีรัม PSA (Prostate Specific Antigen) และอาการ IPSS (International Prostatic Symptom Score) และหาความสัมพันธ์เชิงสถิติ*

ผลการศึกษา: *พบว่าเมื่อได้รับฮอร์โมน testosterone ต่อมลูกหมากส่วน central gland มีขนาดเพิ่มขึ้นอย่างมีนัยสำคัญทางสถิติ ($p = 0.02$) ในช่วงเวลาติดตามเฉลี่ยประมาณ 6 เดือนครึ่ง สำหรับปริมาณหลอดเลือดที่เข้าไปในต่อมลูกหมากในทุกส่วนของต่อมลูกหมากมีปริมาณเพิ่มขึ้นอย่างมีนัยสำคัญทางสถิติเช่นกัน ในส่วนข้อมูลทางคลินิกพบว่าเมื่อได้รับฮอร์โมน ระดับซีรัมเทสโทสเตอโรน ค่า PSA และ PSA density มีค่าสูงขึ้นหลังได้ฮอร์โมน และยังพบความสัมพันธ์ระหว่างขนาดของต่อมลูกหมากที่เพิ่มขึ้นกับค่า PSA ที่สูงขึ้นด้วย ในทางตรงข้าม ค่า IPSS ลดลงอย่างมีนัยสำคัญทางสถิติ ($p < 0.001$)*

สรุป: *ในผู้ป่วยที่ได้รับฮอร์โมนเทสโทสเตอโรนเสริม จะมีขนาดของต่อมลูกหมากใหญ่ขึ้น โดยเฉพาะส่วน central gland ในช่วง 6 เดือนแรกหลังการรักษา และพบว่ามีความสัมพันธ์กับการเพิ่มขึ้นของค่า PSA นอกจากนี้ยังพบว่ามีการเพิ่มขึ้นของหลอดเลือดเข้าไปในต่อมลูกหมาก*