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ORIGINAL ARTICLE

Sexual Function

# Effect of prolactin on penile erection: a cross-sectional study

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Although elevated prolactin levels have been shown to inhibit penile erection, the relationship between prolactin and erection of the penile tip or base has not been extensively researched. We therefore investigated the prolactin's effects on erection of the penile tip and base, with a cross-sectional study of 135 patients with erectile dysfunction, based on scores of  $\leq 21$  on the International Index of Erectile Function-5. All patients were tested for nocturnal penile tumescence, blood pressure, serum glucose, total cholesterol, triglyceride, high-density lipoprotein, low-density lipoprotein, luteinizing hormone, follicle-stimulating hormone, prolactin, estradiol, testosterone, and progesterone. Univariate and multivariate analyses were used to assess the associations between prolactin levels and erection at the penile tip and base. We found no obvious relationship between erection time at penile tip and prolactin levels, but observed a negative correlation between base erection time and prolactin level (hazard ratio:  $-2.68$ ; 95% confidence interval [CI]:  $-5.13$ – $0.22$ ). With increasing prolactin concentration, multivariate analysis showed obvious reduction in base erection time among patients with normal Rigiscan results (hazard ratio:  $-3.10$ ; 95% CI:  $-7.96$ – $1.77$ ;  $P < 0.05$ ). Our data indicate that prolactin inhibits penile erection, particularly at the penile base. In addition, when the effective erection time of the penile base lasts longer than 10 min, prolactin has a more obvious inhibitory effect on penile base erection.

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## INTRODUCTION

Erectile dysfunction (ED) is defined as the recurrent or consistent inability to achieve and maintain a sufficient erection to permit satisfactory sexual intercourse.<sup>1</sup> A meta-analysis consortium study reported that ED was present in nearly 17% of all European males in 2004 and that it will affect 322 million European males by 2025.<sup>2,3</sup>

ED may result from psychological, neurologic, hormonal, arterial, or cavernosal impairment or from a combination of these factors.<sup>4,5</sup> This study was performed to investigate the relationship between prolactin (PRL) and ED.

PRL is a 23 kDa-polypeptide hormone secreted by pituitary lactotroph cells, under negative dopaminergic and positive serotonergic control.<sup>6–9</sup> Unlike other hormones secreted by the anterior pituitary gland, PRL secretion is controlled primarily by inhibition from the hypothalamus and is not subject to negative feedback, directly or indirectly, by peripheral hormones. It self-inhibits by a countercurrent flow in the hypophyseal pituitary portal system, which initiates secretion of hypothalamic dopamine and inhibits pulsatile secretion of gonadotropin-releasing hormone (GnRH).<sup>10</sup>

Previous studies have shown that elevated PRL inhibits penile erection.<sup>11–14</sup> However, few studies have shown a relationship between PRL and erection of the penile tip or penile base. The potential relationship between ED and PRL led us to investigate the relationship between PRL levels and erections of the penile tip and penile base.

## PATIENTS AND METHODS

This study included 135 patients with ED, based on scores of  $\leq 21$  on the International Index of Erectile Function-5 from January 2012 to November 2017 at our Andrology Clinic at the Department of Urology in the Second Hospital of Shandong University, Jinan, China. This clinical study was approved by the Institutional Ethics Committee of the Second Hospital of Shandong University. All participants gave written informed consent to participate in the study. Men were included in the study if they had ED longer than 3 months' duration. We excluded patients who had (a) a history of neurologic disease, (b) genital or spinal cord injuries, (c) morbid obesity, (d) use of drugs that affect erectile function, (e) severe heart disease, (f) bleeding disorders, (g) penile fibrosis, (h) hypertension, (i) diabetes, (j) hypogonadism, and/or (k) abnormal androgen profile.

Thorough ED histories were taken from all patients in this study. They also received general, genital, neuromonitoring (*i.e.*, nerve assessment), ultrasonography (*i.e.*, arterial and venous assessment), and urologic physical examinations, blood analyses that included serum glucose, total cholesterol (TC), triglyceride (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), luteinizing hormone (LH), follicle-stimulating hormone (FSH), PRL, progesterone (P), estradiol (E), and testosterone (T). The assays were performed at the laboratory of the Second Hospital of Shandong University, using a biochemical analyzer (Modular Analytics, Roche, Mannheim, Germany).

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Nocturnal penile tumescence tests were performed in all patients using the Rigiscan device (Timm Medical Technologies, Inc., Eden Prairie, MN, USA).<sup>15–17</sup> To reduce interference with sleep quality, the device was applied to each patient's penis for 3 consecutive nights; changes in penile tumescence and radial rigidity were recorded throughout the duration of each night. To ensure accurate readings, patients were asked to avoid insomnia, caffeine, or alcohol intake and to evacuate their bladders before going to sleep. After each monitoring period, all data were transferred to a personal computer and analyzed by Rigiscan device (Timm Medical Technologies, Inc., Eden Prairie, MN, USA). The software recognized an erectile event if there was a 20% increase in loop circumference of  $\geq 3$  min in duration.<sup>18</sup> We recorded erection time and rigidity. All patients had normal nocturnal erectile activity, which comprised at least one erection with rigidity of  $\geq 60\%$  at penile tip or base, lasting longer than 10 min as documented by the Rigiscan. We considered rigidity of  $\geq 60\%$  to be an effective erection.

For both tip and base erections, we chose the recorded effective erection with the longest duration, and then divided participants into those whose longest-lasting effective erection was at the tip (tip group) and those whose longest-lasting effective erection was at the base (base group). We then divided the base group into two subgroups: those whose effective erection time was  $>10$  min (Group 1) and those whose effective erection time was  $\leq 10$  min (Group 2). All research subjects were ED patients, including both organic ED (for which a physical cause could be found) and psychological ED (for which no physical cause could be found). We supposed that, if after all examinations (including the Rigiscan), we found no physical abnormality indicative of organic ED, then the patient's ED was psychological in nature.

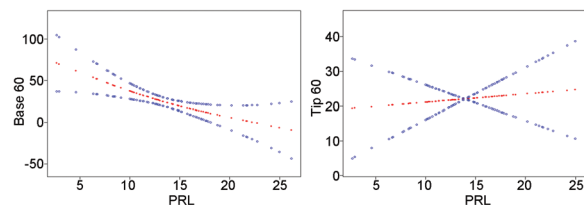
Statistical analyses were performed using Empower Stats software, version 2.18.5 (X&Y Solutions Inc., Boston, MA, USA). Baseline characteristics were presented as mean  $\pm$  standard deviation (s.d.). We conducted regression analysis to fit the smoothing curve. Univariate analyses were performed to determine the significance of variables. Subsequently, multivariate analyses were performed to determine whether PRL levels were independently associated with tip or base erections after adjusting for likely confounders, including age, systolic blood pressure (BP), diastolic BP, blood glucose, total cholesterol, HDL, LDL, FSH, E, LH, or P.

## RESULTS

Basic characteristics of all 135 patients are shown in **Table 1**. Their mean age was  $33.97 \pm 10.70$  years old. Their mean concentrations of PRL and T were  $14.43 \pm 6.63 \mu\text{g l}^{-1}$  and  $4.58 \pm 1.52 \mu\text{g l}^{-1}$ , respectively. Univariate analysis showed a stronger correlation between PRL level and Base 60 (hazard ratio [HR]:  $-0.94$ , 95% confidence interval [CI]:  $-1.88$ – $0$ ) than that between PRL level and Tip 60 (HR:  $-0.48$ , 95% CI:  $-1.56$ – $0.59$ ).

The smoothing curve showed that Tip 60 increased linearly with PRL (**Figure 1**). Base 60 was inversely correlated with PRL level. After adjusting for age, systolic BP, diastolic BP, blood glucose, TC, HDL, LDL, FSH, E, T, LH, P, and total tip duration (the total time of penile tip erection), multivariate analyses showed that a unit increase in PRL was associated with an increase of 0.46 min (95% CI:  $-0.80$ – $1.72$ ) in Tip 60 (**Table 2**). After adjusting for age, systolic BP, diastolic BP, blood glucose, TC, TG, HDL, LDL, FSH, E, T, LH, and P, a unit increase in PRL was associated with a decrease of 2.68 min (95% CI:  $-5.13$ – $-0.22$ ) in Base 60 ( $P < 0.05$ ; **Table 3**). We found no obvious relationship between penile tip erection time and PRL level, but observed a negative correlation between penile base erection time and PRL level (HR:  $-2.68$ ; 95% CI:  $-5.13$ – $-0.22$ ).

Basic characteristics of patients in Groups 1 and 2 are shown in **Supplementary Table 1**. There were 55 patients in Group 1 and 80 patients in Group 2. The mean age of patients in Group 1 ( $33.91 \pm 11.78$  years) was similar to that of patients in Group 2 ( $34.01 \pm 9.96$  years). Patients in Group 1 had a higher mean PRL level ( $14.47 \pm 5.82 \mu\text{g l}^{-1}$ ) than did patients in Group 2



**Figure 1:** Smoothing curve of Tip 60 or Base 60 by PRL. PRL: prolactin; Tip 60: the time penile tip rigidity  $>60\%$ ; Base 60: the time penile base rigidity  $>60\%$ .

**Table 1: General characteristics of all patients and univariate analysis result**

Characteristics	Value, mean $\pm$ s.d.	Tip 60, HR (95% CI)	Base 60, HR (95% CI)
Age (year)	33.97 $\pm$ 10.70	-0.18 (-0.85–0.49)	-0.04 (-0.63–0.55)
Systolic (mmHg)	131.38 $\pm$ 14.40	0.03 (-0.55–0.61)	-0.15 (-0.70–0.39)
Diastolic (mmHg)	83.01 $\pm$ 14.20	-0.47 (-1.06–0.11)	-0.14 (-0.69–0.41)
Blood glucose (mmol l <sup>-1</sup> )	4.85 $\pm$ 0.97	-4.65 (-11.67–2.37)	1.73 (-4.87–8.33)
TC (mmol l <sup>-1</sup> )	4.13 $\pm$ 1.00	-3.97 (-11.43–3.48)	0.74 (-6.13–7.62)
TG (mmol l <sup>-1</sup> )	1.23 $\pm$ 0.71	-1.65 (-12.38–9.08)	0.44 (-9.41–10.29)
HDL (mmol l <sup>-1</sup> )	1.14 $\pm$ 0.28	2.58 (-24.54–29.71)	16.49 (-8.25–41.24)
LDL (mmol l <sup>-1</sup> )	2.76 $\pm$ 3.19	-0.09 (-2.44–2.26)	-0.71 (-2.87–1.44)
E (pg ml <sup>-1</sup> )	56.62 $\pm$ 26.17	0.03 (-0.25–0.30)	-0.03 (-0.27–0.21)
FSH (mIU ml <sup>-1</sup> )	5.52 $\pm$ 4.43	1.11 (-0.49–2.71)	-1.12 (-2.53–0.29)
LH (mIU ml <sup>-1</sup> )	5.17 $\pm$ 6.10	0.20 (-0.97–1.37)	-0.31 (-1.34–0.73)
PRL (ng ml <sup>-1</sup> )	14.43 $\pm$ 6.63	-0.48 (-1.56–0.59)	-0.94 (-1.88–0.00)
T (ng ml <sup>-1</sup> )	4.58 $\pm$ 1.52	-0.81 (-5.51–3.88)	0.36 (-3.79–4.52)
P (ng ml <sup>-1</sup> )	0.96 $\pm$ 0.54	-7.23 (-20.33–5.87)	2.87 (-8.78–14.51)
Total tip duration (min)	59.67 $\pm$ 88.88	0.29 (0.23–0.36)	–
Total base duration (min)	69.03 $\pm$ 97.70	–	-0.02 (-0.09–0.04)

Tip 60: the time penile tip rigidity  $>60\%$ ; Base 60: the time penile base rigidity  $>60\%$ ; TC: total cholesterol; TG: triglyceride; HDL: high-density lipoprotein cholesterol; LDL: low-density lipoprotein; E: estradiol; FSH: follicle stimulating hormone; LH: luteinizing hormone; PRL: prolactin; T: testosterone; P: progesterone; HR: hazard ratio; CI: confidence interval; –: no significance; s.d.: standard deviation

(14.40 ± 7.31 µg l<sup>-1</sup>). Base 60 in Group 2 was much higher than that in Group 1 (40.72 ± 41.65 min versus 2.54 ± 2.75 min). Group 1 had higher mean blood glucose, TC, TG, LDL, and FSH levels than Group 2; Group 1 had lower mean HDL, E, LH, T, and P levels than Group 2.

Univariate analysis showed a stronger correlation between PRL levels and Group 2 (HR: -0.93, 95% CI: -2.24-0.38) than for Group 1 (HR: -0.03, 95% CI: -0.15-0.09) (*P* < 0.05). Multivariate analysis result showed that, for each unit increase in PRL level, mean erection duration decreased by 0.08 min (95% CI: -0.30-0.13) in Group 1, and by 2.31 min (95% CI: -5.58-0.96) in Group 2, after adjusting for age, systolic BP, diastolic BP, blood glucose, TC, and TG. For each unit increase in PRL level, mean erection duration decreased by 0.07 min (95% CI: -0.35-0.21) in Group 1, and by 3.10 min (95% CI: -7.96-1.77) in Group 2, after adjusting for age, systolic BP, diastolic BP, blood glucose, TC, HDL, LDL, FSH, E, T, LH, and P (*P* < 0.05; **Table 4** and **5**).

**DISCUSSION**

ED is a common condition which interferes with quality of life of affected persons. The World Health Organization states that sexual well-being is essential to the physical and emotional health of individuals, couples, and families and the social and economic improvement of countries.<sup>19</sup>

Penile erection is a neurovascular event modulated by psychological factors and hormonal status. When sexual stimulation, the cavernous nerve terminals release the neurotransmitters and the endothelial cells in the penile release the relaxing factors, resulting in the relaxation of penile vascular smooth muscle and a several-fold increase in penile blood flow. At the same time, relaxation of the trabecular smooth muscle increases the compliance of the sinusoids, facilitating rapid filling and expansion of the sinusoidal system. The subtunical venular plexuses are thus compressed between the trabeculae and the tunica albuginea, resulting in almost total occlusion of venous outflow.<sup>20,21</sup> These events trap the blood within the corpora cavernosa and raise the penis from a dependent position to an erect position, with an intracavernous pressure of approximately 100 mmHg (the phase of full erection).<sup>4</sup> PRL secretion is under the inhibitory control by hypothalamic dopamine, and dopamine secretion is influenced by PRL release.<sup>22</sup> PRL has been suggested to have a role in modulating sexual behavior and activity, through central or peripheral mechanisms. The central mechanisms are likely mediated by the dopaminergic system, which regulates both PRL secretion and sexual function. Notably, dopamine is a major modulator of sexual function;<sup>23,24</sup> moreover, PRL may influence erectile function by modulating dopaminergic function at specific brain sites.<sup>25-28</sup> The mutual interaction between PRL and dopamine, together with the well-known effects of PRL on the hypothalamic-pituitary-gonadal axis, supports the role of PRL as a hormone that regulates sexual function. Elevated PRL is associated with impairment of the hypothalamic-pituitary-gonadal axis.<sup>29</sup> In the central nervous system, hyperprolactinemia inhibits centers that control sexual desire and erection;<sup>30</sup> experiments in mouse models have suggested that inhibition of erectile function occurs through the induction of hyperprolactinemia at a supraspinal level within the central nervous system.<sup>12</sup> Under physiological conditions, PRL specifically binds to Leydig cells and promotes secretion of T by interstitial cells through downstream action of LH; moreover, it acts on the epididymis and prostate to maintain the functionality of the accessory gonads and promote sperm production. Androgen can indirectly promote penile erection by mechanisms such as enhancing sexual desire and sexual arousal; moreover, it plays an important role in maintaining normal erectile tissue structure of the penis. T deficiency can lead to reduction

**Table 2: Multiple regression analysis result of the tip**

Exposure	Nonadjusted	Adjust 1	Adjust 2
PRL, HR (95% CI)	-0.48 (-1.56-0.59)	-0.59 (-2.40-1.23)	0.46 (-0.80-1.72)
<i>P</i>	0.056	<0.05	0.132

Result variable: the time penile tip rigidity >60%; exposure variable: PRL. Adjust 1 model adjusts for: age, systolic, and diastolic; Adjust 2 model adjusts for: age, systolic, diastolic, blood glucose, TC, HDL, LDL, FSH, E, T, LH, P, and total tip duration. TC: total cholesterol; HDL: high-density lipoprotein cholesterol; LDL: low-density lipoprotein; E: estradiol; FSH: follicle stimulating hormone; LH: luteinizing hormone; PRL: prolactin; T: testosterone; P: progesterone; HR: hazard ratio; CI: confidence interval

**Table 3: Multiple regression analysis result of the base**

Exposure	Nonadjusted	Adjust 1	Adjust 2
PRL, HR (95% CI)	-0.94 (-1.88-0.00)	-2.65 (-4.64-0.66)	-2.68 (-5.13-0.22)
<i>P</i>	<0.05	<0.05	<0.05

Result variable: the time penile base rigidity >60%; exposure variable: PRL. Adjust 1 model adjusts for: age, systolic, diastolic, blood glucose, TC, and TG; Adjust 2 model adjusts for: age, systolic, diastolic, blood glucose TC, TG, HDL, LDL, FSH, E, T, LH, and P. TC: total cholesterol; TG: triglyceride; HDL: high-density lipoprotein cholesterol; LDL: low-density lipoprotein; E: estradiol; FSH: follicle stimulating hormone; LH: luteinizing hormone; PRL: prolactin; T: testosterone; P: progesterone; HR: hazard ratio; CI: confidence interval

**Table 4: Univariate analysis result of the Group 1 and Group 2**

Characteristics	Group 1, HR (95% CI)	Group 2, HR (95% CI)	<i>P</i>
Systolic (mmHg)	0.01 (-0.03-0.06)	-0.28 (-1.26-0.70)	0.651
Diastolic (mmHg)	-0.01 (-0.06-0.03)	-0.62 (-1.64-0.40)	0.309
Blood glucose (mmol l <sup>-1</sup> )	-0.88 (-1.69-0.08)	5.26 (-3.84-14.37)	0.281
TC (mmol l <sup>-1</sup> )	-0.28 (-1.11-0.55)	3.12 (-6.46-12.70)	0.539
TG (mmol l <sup>-1</sup> )	-1.18 (-2.44-0.08)	1.83 (-11.23-14.88)	0.833
HDL (mmol l <sup>-1</sup> )	1.67 (-1.73-5.08)	17.21 (-15.37-49.80)	0.251
LDL (mmol l <sup>-1</sup> )	-0.07 (-0.23-0.09)	0.08 (-10.49-10.64)	0.949
E (pg ml <sup>-1</sup> )	-0.01 (-0.03-0.02)	-0.09 (-0.46-0.27)	0.617
FSH (mIU ml <sup>-1</sup> )	0.02 (-0.11-0.15)	-0.91 (-3.84-2.02)	0.678
LH (mIU ml <sup>-1</sup> )	-0.01 (-0.25-0.24)	-0.51 (-1.73-0.71)	0.316
PRL (ng ml <sup>-1</sup> )	-0.03 (-0.15-0.09)	-0.93 (-2.24-0.38)	<0.05
T (ng ml <sup>-1</sup> )	-0.10 (-0.65-0.44)	0.02 (-5.65-5.69)	0.735
P (ng ml <sup>-1</sup> )	0.99 (-0.52-2.50)	-4.94 (-21.12-11.24)	0.563

TC: total cholesterol; TG: triglyceride; HDL: high-density lipoprotein cholesterol; LDL: low-density lipoprotein; E: estradiol; FSH: follicle stimulating hormone; LH: luteinizing hormone; PRL: prolactin; T: testosterone; P: progesterone; HR: hazard ratio; CI: confidence interval

**Table 5: Multivariate analysis result of the base**

Model	Group 1, HR (95% CI)	Group 2, HR (95% CI)	<i>P</i>
Nonadjusted	-0.03 (-0.15-0.09)	-0.93 (-2.24-0.38)	<0.05
Adjust 1	-0.08 (-0.30-0.13)	-2.31 (-5.58-0.96)	<0.05
Adjust 2	-0.07 (-0.35-0.21)	-3.10 (-7.96-1.77)	<0.05

Result variable: the time penile base rigidity >60%; exposure variable: PRL. Adjust 1 model adjusts for: age, systolic, diastolic, blood glucose, TC, and TG; Adjust 2 model adjusts for: age, systolic, diastolic, blood glucose, TC, HDL, LDL, FSH, E, T, LH, and P. TC: total cholesterol; TG: triglyceride; HDL: high-density lipoprotein cholesterol; LDL: low-density lipoprotein; E: estradiol; FSH: follicle stimulating hormone; LH: luteinizing hormone; PRL: prolactin; T: testosterone; P: progesterone; HR: hazard ratio; CI: confidence interval

of trabecular smooth muscle, increased extracellular matrix, deposition of subadhesive fat cells, and other physiological problems. During the erection process, the above changes in the penile tissue may cause venous leak due to venous occlusion, resulting in erectile dysfunction. Excessive PRL can inhibit the release of FSH and LH through the central nervous system, thereby reducing the synthesis and secretion of sex hormones, which affects sperm maturation. In addition, excessive PRL destroys the synergistic effect of gonadotropin, which directly affects



gonadotropin, as well as the responsiveness of accessory gonads to gonadotropin; it also affects the metabolism, motion, and capacitation of sperm, which can reduce male gonad function. Peripherally, chronic hyperprolactinemia has been found to arrest penile erection via direct action at the vascular level in the penile corpus cavernosum of dogs.<sup>13</sup>

Elevated PRL alters the balance among neurotransmitters, neuropeptides, and hormones involved in libido and erection, affecting dopaminergic tone. An imbalance is generated between dopamine which stimulates sexual function, and serotonin which inhibits sexual function. At the neuroendocrine level, elevated PRL decreases GnRH expression, interferes with GnRH action, inhibits gonadotrophin secretion, reduces T to dihydrotestosterone conversion, and decreases central dopamine action.<sup>11,31-33</sup> Some data in animal models have associated acute increases in serum PRL levels with arrested penile erection, probably via inhibition of smooth muscle relaxation of the penile corpus cavernosum.<sup>13,14</sup>

Past studies demonstrated that PRL has a negative effect on erectile function.<sup>11,12</sup> However, to our knowledge, few studies have shown a relationship between PRL and erections at the penile tip or penile base. In addition, there are no uniform standards for normal erections, as determined by Rigiscan analysis. In our present study, univariate analyses confirmed that PRL affected male erectile function. However, we have shown, for the first time, that PRL levels are not strongly associated with penile tip erection duration, and that they are inversely related to penile base erection duration (**Figure 1**). Multivariate analysis showed that PRL level was independently associated with Tip 60 or Base 60, after adjusting for some confounders. Therefore, we concluded that the penile base was more meaningful than the penile tip in assessing the effect of PRL on erectile function, and we performed further analysis of the base group.

By study the Group 1 and Group 2 in Base 60, we could know that the effects of prolactin on the same position of the penile were different as the effective erection changes. In univariate analysis and multivariate analysis, Group 2 showed a greater change than Group 1 in Base 60 when PRL increased. We supposed that the longer the effective erection lasted, the more obvious the effects of prolactin was. We would do further experiments to verify this hypothesis in the future.

The study showed that PRL had a greater effect on the penile base than the penile tip; additionally, there was a more obvious inhibitory effect on base erections when the effective erection lasted longer than 10 min. We concluded that in patients whose effective erections lasted longer than 10 min, the cause of ED was not organic, but psychological.

We suggest that the differences between the penile tip and penile base should be considered when we study the relationship between penile erectile dysfunction and PRL using Rigiscan analysis; base data comprise a more reliable reference set, which will enable more effective and more accurate research.

This study had several limitations. First, there were relatively few subjects and a low number of variables, which might have affected our results. In addition, normal PRL ranges might vary slightly among different laboratory machines, and should therefore be adjusted on the basis of these machines. A series of comprehensive blinded validation studies are warranted to confirm the clinical effects of PRL.

## CONCLUSIONS

In conclusion, we confirmed that PRL inhibits erectile function. We also found that the penile base is more sensitive to PRL than the penile tip. In addition, for penile base effective erections that last longer than 10 min, PRL has a more obvious inhibitory effect on penile base erection.

## AUTHOR CONTRIBUTIONS

STZ and YG designed this study; ZHX performed the statistical analysis and drafted the manuscript; and DP, TYL, MZY, JYZ, SJ, and XSW collected the data and helped design the manuscript. All authors read and approved the final manuscript.

## COMPETING INTERESTS

All authors declared no competing interests.

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Supplementary Information is linked to the online version of the paper on the *Asian Journal of Andrology* website.

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**Supplementary Table 1: General characteristics of the patients in Group 1 and Group 2**

<i>Group</i>	<i>1</i>	<i>2</i>	<i>P</i>
<i>n</i>	55	80	
Age	33.91±11.78	34.01±9.96	0.231
Systolic	131.93±16.61	130.94±12.51	0.749
Diastolic	81.72±16.72	84.04±11.89	0.445
Blood glucose	4.93±0.89	4.79±1.02	0.411
TC	4.21±0.98	4.08±1.02	0.484
TG	1.25±0.64	1.22±0.75	0.831
HDL	1.12±0.24	1.16±0.30	0.549
LDL	3.23±5.07	2.47±0.93	0.212
E	55.49±27.52	57.40±25.35	0.678
FSH	6.60±5.68	4.78±3.15	<0.05
LH	4.81±2.99	5.42±7.54	0.571
PRL	15.34±6.06	13.81±6.97	<0.05
T	4.52±1.35	4.62±1.63	0.721
P	0.85±0.48	1.04±0.57	0.054
Base 60	2.54±2.75	40.72±41.65	<0.01

TC: total cholesterol; TG: triglyceride; HDL: high-density lipoprotein cholesterol; LDL: low-density lipoprotein; E: estradiol; FSH: follicle stimulating hormone; LH: luteinizing hormone; PRL: prolactin; T: testosterone; P: progesterone; Base 60: the time penile base rigidity >60%