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Original Research

Association between testosterone levels and RigiScan parameters of patients with erectile dysfunction

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Abstract

Background: It is difficult to diagnose hypogonadism because of the lack of objective assessments of erectile dysfunction (ED), which is caused by hypogonadism.
Aim: To provide a new approach for diagnosing hypogonadism, this study evaluated the efficacy of nocturnal penile tumescence and rigidity (NPTR) testing with RigiScan for patients with ED with and without hypogonadism.
Methods: From June 2021 to February 2023, 133 patients with ED (62 with hypogonadism and 71 without) underwent NPTR testing at the Department of Andrology. A detailed history of all participants was obtained. All participants also underwent a physical examination, sex hormone testing, and ultrasound examination of the cavernous vessels of the penis.
Outcomes: Patient characteristics, sex hormone serum levels, and RigiScan Plus data of NPTR testing of patients with ED were obtained and evaluated.
Results: Between the groups, there were no significant differences in age, body mass index, or erectile function score or in the prevalence of smoking, drinking, diabetes, hypertension, and hyperlipidemia. RigiScan data revealed differences in erection episodes per night, average event rigidity, erection durations, and percentage of tumescence greater than baseline, which were significantly lower in the testosterone-deficient group than in the normal testosterone group. The average event rigidity of the tip displayed the largest area under the curve value, with a sensitivity of 67.6%, a specificity of 85.5%, and a cutoff value of 52.50.
Clinical Implications: Our findings may allow appropriate patients to receive testosterone replacement therapy, which has been shown to be an effective treatment for hypogonadism.
Strengths and Limitations: This is the first study of its kind to perform a comprehensive review of the association between hypogonadism and RigiScan parameters. This study was limited by its small sample size.
Conclusion: RigiScan parameters of patients with ED and testosterone deficiency were significantly lower than those of patients with normal testosterone; therefore, RigiScan is useful for the differential diagnosis of patients with ED caused by hypogonadism.

Keywords: RigiScan; erectile dysfunction; testosterone; hypogonadism.

Introduction

Male hypogonadism is a common endocrine disorder whereby the male gonads become less functional, resulting in the reduced production of testosterone. Therefore, male hypogonadism is also known as *testosterone deficiency*. The main manifestations of male hypogonadism include decreased sexual desire, erectile dysfunction (ED), and reduced frequency of nocturnal erections. As a result of low testosterone levels, patients with hypogonadism often experience depression, fatigue, obesity, and other systemic symptoms.[1] Testosterone deficiency can be caused by many factors. Primary hypogonadism is more common in patients with Klinefelter's syndrome and testicular dysplasia, whereas secondary hypogonadism is more common in patients with type 2 diabetes, metabolic syndrome, and any dysfunction of the hypothalamic-pituitary-testis gonadal axis.[2] The diagnosis of hypogonadism mainly depends on the signs and symptoms caused by low testosterone[3] and is made when the patient experiences >2 instances of significantly lower-than-normal serum testosterone levels (8 nmol/L) during the morning.[4]

At present, the diagnosis of ED must be made in combination with a comprehensive assessment of the medical history, physical examination, score on the 5-item International Index of Erectile Function (IIEF-5), and sex hormone levels,[5] which are difficult to quantify objectively. The development of RigiScan has allowed this objective evidence to be obtained. Various studies have shown that monitoring nocturnal penile tumescence and rigidity (NPTR) plays an important role in the diagnosis of male erectile function and is the gold standard method for differentiating psychogenic and organic ED, with sensitivity and specificity of 87% and 88%, respectively.[6] Fisher et al[7] first reported the association between nocturnal erections and rapid eye movement sleep; they found that men experienced 3 to 5 erections of varying degrees during nighttime sleep. Based on these findings, NPTR was combined with RigiScan to record the number and duration of erections and the degree of penis tumescence and rigidity during the nighttime sleep of patients to assess erectile function.

In 1997, Granata et al[8] performed a study of the relationship between the nocturnal erections and testosterone levels of men and found that the number of nocturnal erections and the

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tumescence and rigidity of the penis were significantly lower in men with lower testosterone levels than in controls. However, Buena et al[9] found that when testosterone levels were within the normal range, there was no significant difference in the number of nocturnal erections and their tumescence, rigidity, and duration among male patients with different testosterone levels. As such, NPTR is an important test for the diagnosis of ED, and it can reflect, to a certain extent, the difference in erectile function between patients with testosterone deficiency and those with normal testosterone, thereby improving the diagnosis rate of male hypogonadism.

The primary aim of this study was to evaluate the effect of testosterone levels on RigiScan parameters in patients with ED. The secondary aim was to identify the specific differences in RigiScan parameters between patients with ED who have normal and abnormal testosterone.

Methods

A total of 133 patients with ED (IIEF-5 score <21) were enrolled in the study from June 2021 to February 2023 at the Department of Andrology. All the patients signed the informed consent form. The study was approved by our center's institutional review board. A detailed history was taken from all patients, who also underwent physical examination, sex hormone level testing, ultrasound of the cavernous vessels of the penis, and NPTR testing. Patients with penile abnormalities, penile vascular injury, nerve injury, and serious psychological problems were excluded from the study.

Serum levels of sex hormones (chemiluminescence immunoassay), including testosterone, estradiol, prolactin, and luteinizing hormone, were measured in all patients on an empty stomach and in the morning after fasting (between 7 and 11 AM). Among them, 62 patients with testosterone levels <8 nmol/L underwent repeated serum testosterone measurements to confirm testosterone deficiency. These patients composed the testosterone-deficient group. Inclusion criteria were as follows: testosterone deficiency (testosterone <8 nmol/L), IIEF-5 score ≤21, age >22 years but <70 years, participation in a stable heterosexual relationship for ≥3 months, consent to at least 4 attempts of sexual intercourse every 4 weeks during the trial, and no organic lesions that led to ED (eg, penile abnormalities, penile vascular injury, nerve injury, and serious psychological problem). The remaining 71 patients with normal testosterone levels composed the normal testosterone group. Inclusion criteria were as follows: no testosterone deficiency (>8 nmol/L), IIEF-5 score ≤21, age >22 years but <70 years, participation in a stable heterosexual relationship ≥3 months, consent to at least 4 attempts of sexual intercourse every 4 weeks during the trial, and no organic lesions that led to ED (eg, penile abnormalities, penile vascular injury, nerve injury, and serious psychological problem).

The RigiScan Plus instrument (GOTOP Medicine) was used for NPTR testing of both groups in the hospital under the same conditions. All patients underwent NPTR testing for at least 2 nights (starting at 10 PM and ending at 7 AM the next day) based on the premise that their sleep quality was guaranteed. The following RigiScan data were collected for analysis: the number of nocturnal erections and average event rigidity, duration of erections ≥60% (minutes), baseline circumference (centimeters), average event tumescence (centimeters), and event tumescence of the tip and base greater than baseline (percentage).

Data analysis was performed with SPSS software version 26.0 (IBM Corp). Independent variables (smoking, drinking, diabetes, hypertension, and hyperlipidemia) were analyzed by the chi-square test or Fisher's exact chi-square test. Measurement data are expressed as mean and SD. The measurement data of all participants were tested for normality and homogeneity of variance (age, body mass index [BMI], IIEF-5 score, libido score, medical history, sex hormone levels, and RigiScan data). Independent sample Student's *t*-tests were performed on data conforming to normality. Nonnormal data are expressed as median (IQR), and comparisons were performed with nonparametric tests. $P < .05$ indicated a statistically significant difference. Receiver operating characteristic analysis was performed on RigiScan data to determine the cutoff values, sensitivity, and specificity of relevant variables.

Results

Table 1 shows the baseline characteristics of the patients included in this study. No significant differences were observed in age (mean ± SD; 32.90 ± 7.38 vs 34.65 ± 8.57 years), medical history (7.72 ± 4.88 vs 7.27 ± 5.71 months), BMI (23.50 ± 2.53 vs 24.20 ± 3.04 kg/m^2), and IIEF-5 score (11.89 ± 4.10 vs 11.16 ± 3.83) between the normal testosterone and testosterone-deficient groups, respectively. Additionally, there were no statistically significant differences in other factors that can affect erectile function, such as smoking, drinking, diabetes, hypertension, and hyperlipidemia ($P > .05$). However, the sexual desire scores of the normal testosterone and testosterone-deficient groups were significantly different (6.30 ± 1.31 and 3.77 ± 1.17, respectively; $P < .001$).

Table 2 shows the serum levels of sex hormones in the 2 groups. The mean testosterone level in the normal testosterone group was 15.29 ± 4.55 nmol/L, whereas that of the testosterone-deficient group was 6.99 ± 1.76 nmol/L, showing a significant statistical difference ($P < .001$). Estradiol levels were also significantly higher in the normal testosterone group (145.64 ± 41.88 pmol/L) vs the testosterone-deficient group (131.15 ± 40.16 pmol/L, $P = .044$). In addition, we compared the proportion of testosterone to estradiol of the normal testosterone group (0.111 ± 0.039) with that of the testosterone-deficient group (0.058 ± 0.026, $P < .001$). There were no differences in the luteinizing hormone or prolactin levels between the groups.

Table 3 shows the RigiScan data of patients in the 2 groups after NPTR testing. There were no significant differences in the baseline circumferences of the penile tip and base between the groups. The number of nocturnal erections of the normal testosterone group was 5.13 ± 1.80, which was greater than that of the testosterone-deficient group (3.79 ± 2.03, $P < .001$). The average event rigidity, duration of erections, and tumescence greater than the baseline of the tip and base were all significantly higher in the normal testosterone group than the testosterone-deficient group.

The results of the receiver operating characteristic analysis of the RigiScan data are shown in Table 4. The clinical application value of RigiScan parameters for predicting hypogonadism in patients was determined according to the cutoff value, sensitivity, specificity, and area under the curve of each variable. According to the results of these analyses, the average event rigidity of the tip had the highest diagnostic value for hypogonadism, with an area under the curve of 0.83

Table 1. Characteristics of patients with erectile dysfunction.

	Patients, mean ± SD or No.		
	Normal testosterone (*n* = 71)	Testosterone deficient (*n* = 62)	*P* value
Age, y	32.90 ± 7.38	34.65 ± 8.57	.210
BMI, kg/m^2	23.50 ± 2.53	24.20 ± 3.04	.149
IIEF-5 score	11.89 ± 4.10	11.16 ± 3.83	.295
Libido score	6.30 ± 1.31	3.77 ± 1.17	<.001
History, mo	7.72 ± 4.88	7.27 ± 5.71	.629
Smoking			.875
Yes	11	9	
No	60	53	
Drinking			.787
Yes	18	17	
No	53	45	
Diabetes			.892
Yes	5	4	
No	66	58	
Hypertension			.700
Yes	9	9	
No	62	51	
Hyperlipidemia			.406
Yes	13	15	
No	58	47	

Abbreviations: BMI, body mass index; IIEF-5, 5-item International Index of Erectile Function.

Table 2. Serum levels of sex hormones in patients with erectile dysfunction.

	Serum levels, mean ± SD		
	Normal testosterone (*n* = 71)	Testosterone deficient (*n* = 62)	*P* value
TT, nmol/L	15.29 ± 4.55	6.99 ± 1.76	<.001
E2, pmol/L	145.64 ± 41.88	131.15 ± 40.16	.044
TT/E2	0.111 ± 0.039	0.058 ± 0.026	<.001
PRL, mIU/L	157.82 ± 64.54	175.13 ± 60.93	.118
LH, IU/L	3.74 ± 1.61	3.37 ± 1.84	.218
IGF-1	166.67 ± 35.53	180.68 ± 40.92	.151

Abbreviations: E2, estradiol; IGF-1, insulin-like growth factor 1; LH, luteinizing hormone; PRL, prolactin; TT, testosterone.

Table 3. RigiScan Plus data from nocturnal penile tumescence and rigidity testing.

	RigiScan Plus data, mean ± SD		
	Normal testosterone (*n* = 71)	Testosterone deficient (*n* = 62)	*P* value
Erection episodes/night	5.13 ± 1.80	3.79 ± 2.03	<.001
Tip			
Average event rigidity,%	57.55 ± 11.28	39.42 ± 16.38	<.001
Duration of erections ≥60%, min	12.00(14.0)	3.00(4.3)	<.001
Baseline circumference, cm	5.71 ± 0.57	5.80 ± 0.71	.388
Event tumescence, cm	7.80 ± 1.15	7.11 ± 2.32	.035
Event tumescence >baseline, %	37.41 ± 14.12	29.77 ± 14.99	.003
Base			
Average event rigidity,%	54.10 ± 9.94	39.44 ± 17.34	<.001
Duration of erections ≥60%, min	7.50 ± 14.5	2.25 ± 5.5	<.001
Baseline circumference, cm	6.04 ± 0.55	6.19 ± 0.67	.157
Event tumescence, cm	8.44 ± 1.02	7.75 ± 2.33	.035
Event tumescence >baseline, %	40.37 ± 11.25	33.05 ± 14.66	.001

Table 4. Receiver operating characteristic analysis of RigiScan data.

	Cutoff value	Area under the curve	Sensitivity, %	Specificity, %
Erectile episodes/night	4.0	0.67	81.7	43.5
Average event rigidity, %				
Tip	52.50	0.83	67.6	85.5
Base	49.50	0.77	70.4	75.8
Duration of erections ≥60%, min				
Tip	6.25	0.75	66.2	77.4
Base	3.25	0.76	77.5	69.4
Event tumescence >baseline, %				
Tip	19.5	0.63	95.8	29.0
Base	39.5	0.67	51.7	75.3

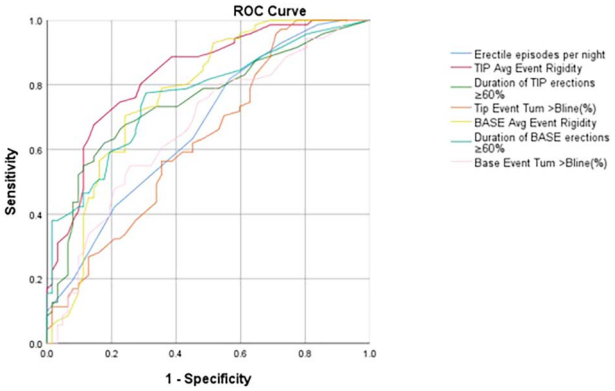

Figure 1. The ROC curve of RigiScan parameter.

(Figure 1). When the cutoff value was 52.50, the sensitivity was 67.6% and the specificity was 85.5%.

Discussion

Male ED is a common disease that affects the quality of life of patients. This condition, which mainly affects middle-aged and older men, is also becoming increasingly common in young individuals.[10] The incidence of ED among men aged >40 years can reach 9%[11]; however, this incidence ranges from 20% to 40% among men aged >60 years. Since the efficacy of phosphodiesterase 5 (PDE-5) inhibitors for the treatment of ED has been discovered, these drugs have been widely used. A previous study showed that the clinical effectiveness rate of PDE-5 inhibitors is approximately 70%. Yet, for many patients, the use of PDE-5 inhibitors alone does not achieve satisfactory results; therefore, further research of the diagnosis and treatment of this condition is required.

The causes of ED in men are complex. Common causes are nerve injury, endocrine abnormalities (eg, hypogonadism, diabetes, and hyperprolactin), vasogenic (eg, hypertension, hyperlipidemia, diabetes and other causes of microvascular lesions, and arteriovenous fistula), drug use (eg, antihypertensive drugs, antiandrogenic drugs, and sedatives), and lifestyle factors (eg, smoking and drinking).[12] No patients with arteriovenous fistula or nerve injury were included in this study. The 2 groups of patients differed only in levels of sex hormones and showed no differences in angiogenic, lifestyle, or other factors. As such, the purpose of this study was to investigate the association between testosterone levels and RigiScan parameters.

ED caused by hypogonadism is the result of the abnormal structure of erect penis tissue caused by testosterone deficiency and the decreased expression of the nitric oxide receptor, which leads to a reduction in the diastolic function of penile vascular smooth muscle.[13] In clinical practice, exogenous testosterone supplements are therefore often administered to restore normal testosterone levels and eliminate the physiologic changes and clinical symptoms associated with testosterone deficiency. In a number of experimental studies, testosterone replacement therapy has been proven to be effective in improving the sexual desire and erectile function in patients with hypogonadism. Snyder et al[14] performed testosterone supplementation for 790 patients aged >65 years with hypogonadism, which increased the testosterone level to that of healthy men aged 19 to 40 years. According to sexual and physical function assessments and vitality tests, sexual frequency, libido score, and erectile function were significantly improved, and the increased testosterone levels were associated with improvements in mood and depression. Shamloul et al[15] administered testosterone supplementation to older male patients with partial androgen deficiency insensitive to sildenafil, which resulted in significantly improved IIEF-5 scores. Thus, the correct diagnosis of hypogonadism in patients with ED allows testosterone supplementation to be administered, which effectively improves their erectile function.

The diagnosis of hypogonadism requires >2 morning measurements of serum testosterone <8 nmol/L and the presence of ED, loss of libido, and other generalized symptoms associated with low testosterone. The guidelines of the European Society of Urology recommend that patients with ED undergo morning laboratory tests to assess total testosterone, blood glucose, and blood lipid levels after a detailed medical history has been obtained and after psychological and physical assessments have been performed.[16] Therefore, for patients with ED and testosterone levels <8 nmol/L, further objective assessments of erectile function would aid the diagnosis of hypogonadism.

NPTR, as the preferred objective test for the assessment of ED in patients, is the gold standard for distinguishing psychogenic ED from organic ED. In combination with RigiScan software, NPTR testing has a certain value in the identification of arteriovenous[17] and diabetic ED,[18] exploring the influence of age on erectile function[19] and evaluating the efficacy of PDE-5 inhibitors for patients with ED.[20] Evaluating the

particular value of RigiScan for the diagnosis of andrologic diseases may provide a basis for further treatment programs.

By comparing the differences in RigiScan parameters between patients with ED caused by hypogonadism and patients with ED and normal testosterone levels, this study explored the association between testosterone levels and RigiScan parameters and analyzed the application value of RigiScan for diagnosing hypogonadism in patients with ED.

In the present study, all patients in the testosterone-deficient group had testosterone levels <8 nmol/L and IIEF-5 scores <21, with an average of 11.16 ± 3.83, meeting the requirements of preliminary diagnosis. According to the results of NPTR, the patients in the testosterone-deficient group did not reach the normal level of 4 to 6 erections per night for >10 minutes.[16] These findings were in line with the results of IIEF-5 scores and confirmed the diagnosis. The IIEF-5 scores of patients with ED and normal testosterone levels were all <21 points, with an average of 11.89 ± 4.10, which was not statistically different from that of the testosterone-deficient group. The NPTR results of the normal testosterone group were also consistent with the diagnosis of ED. To exclude the influence of other factors on the NPTR results, we confirmed that there were differences only in baseline sex hormone levels and libido scores and no significant differences in baseline data such as age, BMI, medical history, smoking, drinking, diabetes, hypertension, and hyperlipidemia between the groups. The libido score is affected by testosterone levels but does not affect nocturnal erections. After other influencing factors were excluded, results of the RigiScan parameter comparison revealed that the NPTR performance of patients with ED and testosterone deficiency was inferior to that of patients with ED and normal testosterone levels. Finally, patients with ED and lower-than-normal testosterone levels had lower RigiScan parameters than patients with normal testosterone levels. Thus, testosterone levels can affect the NPTR test results of patients with ED.

A receiver operating characteristic curve analysis of the RigiScan data was conducted, and it revealed that RigiScan, a noninvasive means of examination, had good value for predicting hypogonadism. The average event rigidity of the tip was the parameter with the highest diagnostic value, with an area under the curve of 0.83. When the cutoff value was 52.50, the sensitivity and specificity were 67.6% and 85.5%, respectively. This suggests that hypogonadism could be excluded as the etiology of ED in patients whose average event rigidity of the tip was >52.50. This result specifies and standardizes the "ED symptoms" of hypogonadism, thereby aiding the clinical diagnosis of this condition.

Interestingly, the average age of patients with hypogonadism enrolled in our study was only 34 years. This average age was different from that at the onset of hypogonadism. As such, we further considered the reasons for this phenomenon. Because of social factors, an increasing number of young individuals have presented to our andrology outpatient department; however, many elderly men with secondary hypogonadism caused by age often ignore their own symptoms. These factors resulted in the low average age of patients who presented to our andrology clinic. Regarding the patients with hypogonadism who were included in our study, we considered only the effects of testosterone on their erections; we did not distinguish between primary and secondary hypogonadism. Thus, a certain proportion of young patients with primary hypogonadism were in the testosterone-deficient group. These 2 factors led to some population bias in our study. Even though strict inclusion criteria were established and the influence of other factors on erectile function was excluded as far as possible, the sample size was small, resulting in limited statistical power. Therefore, this study provides information that could be beneficial to the further exploration of the application of RigiScan in the field of andrology.

Conclusion

Monitoring NPTR via RigiScan can reflect the effect of testosterone levels on RigiScan parameters in patients with ED. In addition, RigiScan parameters can distinguish the erectile status of patients with ED as a result of hypogonadism and those with normal testosterone to some extent, which has a certain reference value for the differential diagnosis of hypogonadism.

Author Contributions

Fu Yuli (Conceptualization, Writing—review & editing, Writing—original draft), Zhang Qi and Liao Zedong (Data curation), Shi Tianhao and Feng Yanfei (Formal analysis), Yu Haojie, Huang Wenjie and Xu Runnan (Methodology)

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Conflicts of interest

None declared.

References

1. Lunenfeld B, Mskhalaya G, Zitzmann M, *et al*. Recommendations on the diagnosis, treatment and monitoring of hypogonadism in men. *Aging Male*. 2015;**18**:5–15.
2. European Association of Urology. *EAU Guidelines on Sexual and Reproductive Health 2022*. European Association of Urology; 2022.
3. Corona G, Goulis DG, Huhtaniemi I, *et al*. European Academy of Andrology (EAA) guidelines on investigation, treatment and monitoring of functional hypogonadism in males: endorsing organization: European Society of Endocrinology. *Andrology*. 2020;**8**: 970–987.
4. Ross A, Bhasin S. Hypogonadism: its prevalence and diagnosis. *Urol Clin North Am*. 2016;**43**:163–176. https://doi.org/10.1016/j.ucl.2016.01.002.
5. Shamloul R, Ghanem H. Erectile dysfunction. *Lancet*. 2013;**381**:153–165.
6. Basar MM, Atan A, Tekdogan UY. New concept parameters of RigiScan in differentiation of vascular erectile dysfunction: is it a useful test? *Int J Urol*. 2001;**8**:686–691.
7. Fisher C, Gross J, Zuch J. Cycle of penile erection synchronous with dreaming (REM) sleep: preliminary report. *Arch Gen Psychiatry*. 1965;**12**:29–45.

8. Granata AR, Rochira V, Lerchl A, Marrama P, Carani C. Relationship between sleep-related erections and testosterone levels in men. *J Androl*. 1997;**18**:522–527.
9. Buena F, Swerdloff RS, Steiner BS, *et al*. Sexual function does not change when serum testosterone levels are pharmacologically varied within the normal male range. *Fertil Steril*. 1993;**59**: 1118–1123.
10. Ludwig W, Phillips M. Organic causes of erectile dysfunction in men under 40. *Urol Int*. 2014;**92**:1–6.
11. Nicolosi A, Moreira ED Jr, Shirai M, Bin Mohd Tambi MI, Glasser DB. Epidemiology of erectile dysfunction in four countries: cross-national study of the prevalence and correlates of erectile dysfunction. *Urology*. 2003;**61**:201–206.
12. Yafi FA, Jenkins L, Albersen M, *et al*. Erectile dysfunction. *Nat Rev Dis Primers*. 2016;**2**(1):16003.
13. Traish AM, Goldstein I, Kim NN. Testosterone and erectile function: from basic research to a new clinical paradigm for managing men with androgen insufficiency and erectile dysfunction. *Eur Urol*. 2007;**52**(1):54–70.
14. Snyder PJ, Bhasin S, Cunningham GR, *et al*. Effects of testosterone treatment in older men. *N Engl J Med*. 2016;**374**(7): 611–624.
15. Shamloul R, Ghanem H, Fahmy I, *et al*. Testosterone therapy can enhance erectile function response to sildenafil in patients with PADAM: a pilot study. *J Sex Med*. 2005;**2**(4): 559–564.
16. Cilurzo P, Canale D, Turchi P, *et al*. The RigiScan system in the diagnosis of male sexual impotence. *Arch Ital Urol Nefrol Androl*. 1992;**64**(suppl 2):81–85.
17. Zhang Y, Zhang Z, Zhang N. Role of RigiScan parameters in differentiation of vascular erectile dysfunction. *Andrologia*. 2020;**52**(10):e13620. https://doi.org/10.1111/and.13620.
18. Bax G, Marin N, Piarulli F, Lamonica M, Bellio F, Fedele D. Rigiscan evaluation of specific nervous impairment in patients with diabetes and erectile disorders. *Diabetes Care*. 1998;**21**(7):1159–1161.
19. Yaman O, Tokatli Z, Ozdiler E, Anafarta K. Effect of aging on quality of nocturnal erections: evaluation with NPTR testing. *Int J Impot Res*. 2004;**16**(2):150–153.
20. Seftel AD. Re: a prospective, randomized, placebo-controlled trial of on-demand vs nightly sildenafil citrate as assessed by RigiScan and the international index of erectile function. *J Urol*. 2016;**196**(2):514–516.