

EVALUATION OF THE EFFECT OF ANDROGEN REPLACEMENT THERAPY USING TRANSDERMAL TESTOSTERONE IN MALES WITH OBESITY AND HYPOGONADISM



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BACKGROUND: Being an indication for androgen replacement therapy, male hypogonadism is often associated with obesity. Therefore, assessing the impact of testosterone replacement therapy (TRT) on obesity is relevant.

AIMS: To evaluate the impact of transdermal TRT on obesity and quality of life (QOL) in males with hypogonadism.

MATERIALS AND METHODS: A comparative, randomized, prospective study enrolled 156 hypogonadal and obese males aged 39 [34; 44] years (the comparison group had no concurrent intervention). Study participants were randomly assigned 1 of the 2 groups by matching appropriate pairs. Group 1 consisted of males receiving transdermal TRT, while Group 2 consisted of patients not receiving androgen therapy but undergoing obesity treatment through diet and lifestyle modification. The observation period was 12 months. A retrospective comparison was also performed on patient subgroups within Group 2, based on whether their hypogonadism was eliminated or persisted. The procedures included collecting patient anamnesis, conducting a physical examination to determine body mass index, administering a questionnaire regarding androgen deficiency symptoms, and assessing total testosterone levels. The Mann–Whitney U-test, Wilcoxon test, and χ^2 with Yates' correction were employed for group comparisons. A p-value below 0.05 was deemed statistically significant. Multiple comparisons were adjusted using the Bonferroni correction.

RESULTS: The groups were comparable regarding the parameters examined at the study's outset. Most of the parameters observed showed statistically significant alterations in both groups 1 and 2, except for hematocrit and hemoglobin levels in group 2. Patients receiving TRT after 12 months from their inclusion into the study had a statistically significantly higher level of total testosterone and lesser severity of androgen deficiency symptoms. When assessing the magnitude of changes in the studied parameters, it was found that patients receiving TRT were characterized by a statistically significantly more pronounced decrease in body weight (mean difference -4.0% (95% CI -5.2; -2.7, $p < 0.001$), a decrease in waist circumference, as well as the severity of androgen deficiency symptoms. In the group of males not receiving TRT, but undergoing obesity treatment through diet and lifestyle modification, 12 months following the commencement of the study, 31 men showed resolution of hypogonadism. Hematocrit elevations above the reference value ($>50\%$) were observed in 23% of men on TRT and 12% of patients without hypogonadism therapy, $p = 0.098$ (χ^2 with Yates' correction). Hemoglobin elevations above the reference value (>160 g/L) were detected in 37% and 15% of males on TRT and without hypogonadism therapy, respectively ($p = 0.003$; χ^2 with Yates' correction). No increases in hematocrit or hemoglobin levels requiring discontinuation of TRT were observed.

CONCLUSIONS: Treatment of hypogonadism using TRT has a positive effect on weight loss in obese males. Furthermore, normalization of testosterone levels leads to a reduction in androgen deficiency symptoms, accompanied by an improvement in quality of life (QOL). Hypogonadism cessation with obesity treatment alone (without the use of TRT) is possible in 41% (95% CI 30.1–53.3) of cases, provided clinically significant weight loss is achieved. Since most respondents achieved normalization of total testosterone levels within the first 6 months, this allows timely initiation of TRT to those patients who have not achieved stable normalization of total testosterone levels within a specified time.

KEYWORDS: hypogonadism; males; testosterone; testosterone replacement therapy (TRT); obesity; mass-spectrometry.

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It was found that at least 16% of men over 18 years old are obese, and 43% of men of the world population are overweight, and the World Health Organization forecasts that the proportion of such people will grow [1]. Available data confirm the close pathogenetic relationship of overweight/obesity with testosterone deficiency [2, 3]. The mechanisms of the negative effect of excess adipose tissue on testicular function are diverse. Thus, according to some researchers, a decrease in the amplitude of luteinizing hormone (LH) release peaks and an increase in their frequency in obesity can lead to desensitization of Leydig cells and to a stimulating effect of gonadotropin [4]. Adipose tissue in obese individuals also shows increased activity of aromatase enzyme converting testosterone to estrogens [5]. An increased level of estrogens, in turn, reduces both the amplitude of pulsed LH secretion and can directly enhance adipogenesis [6]. In addition, obesity significantly changes the release pattern of adipocytokines [7]. Increased leptin secretion in obesity disrupts central signaling and contributes to the development of hypothalamic-pituitary-testicular axis dysfunction [8]. According to a number of publications, hyperleptinemia, typical in obesity, is associated with the occurrence of androgen deficiency [9]. In addition, obesity reduces the production of adiponectin, which is associated with testosterone deficiency [10]. There is also an association of systemic inflammation in obesity with a decrease in testosterone levels [11]. The result of this impact is the development of either normogonadotropic functional potentially reversible hypogonadism with a disturbance of the negative feedback of the pituitary gonad, or organic mixed normogonadotropic irreversible hypogonadism [5, 11]. The main method used to correct hypogonadism is testosterone replacement therapy (TRT) [2]. Therefore, it is relevant to study both the effect of TRT on obesity and the effect of correction of lifestyle and nutrition on the restoration of endogenous testosterone production, which necessitates this study.

AIMS

To evaluate the impact of transdermal TRT on obesity and quality of life (QOL) in males with hypogonadism.

METHODS

Study design

Prospective, comparative (versus no competitive intervention), randomized trial. The study enrolled 156 men aged 39 [34; 44] years who were randomized into 2 groups by matching pairs. Match criteria: body mass index (BMI) with an acceptable difference ± 2 kg/m², waist circumference (RT) with an acceptable difference ± 2 cm. Thus, Group 1 in the study included male subjects receiving transdermal testosterone concurrently with dietary and lifestyle correction (81 subjects), and Group 2 male subjects who were given recommendations only for dietary and lifestyle correction (75 subjects).

Eligibility criteria

Inclusion Criteria: male gender, age 18–65 years, established diagnosis of obesity, established diagnosis of hypogonadism in accordance with clinical guidelines [2, 12].

Exclusion criteria: hypergonadotropic or hypogonadotropic types of hypogonadism whose pathogenesis is not associated with obesity; gender and developmental disorders, cryptorchidism; absence of at least one of the testicles, trauma and/or genital surgery; androgens, anabolic steroids, gonadotropins, antiestrogens, or antiandrogens at the time of the study or in the past, alcoholism or drug addiction; contraindications to transdermal testosterone; diabetes mellitus.

Withdrawal criteria: violations of the study protocol, refusal to participate in the study, development of conditions in which testosterone therapy is contraindicated.

Realization conditions

The study enrolled obese male patients who sought medical treatment at the I.I. Dedov National Medical Research Center of Endocrinology of the Ministry of Health of Russia.

Study duration

The data for the study were collected from November 2023 to January 2026. The follow-up period was 12 months.

Description of the medical intervention

Blood was taken from the ulnar vein in the morning on an empty stomach. Group 1 patients were prescribed transdermal testosterone (testosterone gel 1% — 5 g) AndroGel® (Besins Healthcare RUS). The adequacy of dosing was assessed by determining the total blood testosterone level in the morning on an empty stomach at the 4th hour after application of the drug to the abdominal skin. The dose was considered optimal with a total testosterone level of 12.1 to 25.0 nmol/L. The duration of the intervention was 12 months. All patients were given weight loss recommendations, including hypocaloric nutrition and daily aerobic exercise. The calorie content of the daily diet in kcal was calculated according to the formula recommended by WHO, taking into account age, weight, and physical activity level for men aged 31–60 years: $(0,0484 \times \text{body weight in kg} + 3.653) \times 240$. Since all subjects had low levels of physical activity, a coefficient of 1 was used. To reduce body weight, the value obtained was reduced by 10%. The minimum daily calorie intake was 1800 kcal. If the patient's initial daily calorie consumption was 3000 kcal or more, then it was gradually reduced by 300–500 kcal per week until the calculated individual calorie target was reached. All patients were recommended a fractional meal (at least 4 times a day), so that its main volume fell on the first half of the day, dinner with a calorie content of no more than 15–20% of the daily total and no later than 4 hours before bedtime. The balance of macronutrients was distributed as follows: consumption of fat was reduced to 25–30% of the daily calorie intake, the proportion of proteins was 15–20%, carbohydrates 55–60%. To increase the level of physical activity, all patients were recommended daily walking 10,000 steps and moderate strength loads of 30–45 minutes a day at least 3 times a week. The duration of the lifestyle correction intervention was 12 months.

Primary study outcome

Assessment of weight change in obese men with hypogonadism with TRT versus no androgen therapy.

Secondary study outcomes

Identification of factors influencing the restoration of endogenous testosterone production in men with obesity and hypogonadism having correction of their lifestyle and nutrition.

Subgroup analysis

Retrospective comparison of patient subgroups of Group 2 according to elimination or persistence of hypogonadism.

Outcome registration methods

Along with interview and history collection, a survey was conducted using the Androgen Deficiency Symptom Questionnaire. Physical examination assessed the condition of sexual hair, mammary glands and external genitalia. Total testosterone was determined by high performance liquid chromatography with tandem mass spectrometry (HPLC-MS/MS) on Agilent 1290 Infinity II chromatograph and AB Sciex TripleQuad 5500 mass spectrometer. Hypogonadism was confirmed when total testosterone in blood serum was below 12.1 nmol/L. LH was determined by immunochemiluminescence analysis (ICLA) using Vitros ECI 3600 automatic analyzer (Ortho-Clinical Diagnostics); complete blood count was performed on Sysmex XN-1000 analyzer (Sysmex Corp., Japan).

Ethical review

The study was approved at a meeting of the local ethics committee of the State Research Center of the Russian Federation of the Federal State Budgetary Institution «National Medical Research Center of Endocrinology» of the Ministry of Health of Russia «Mass spectrometric diagnostics

and personalization of the treatment of hypogonadism syndrome in obese men» (minutes No. 20 of 08.11.2023). The presented work is a fragment of the interventional part of the study.

Statistical analysis

Principles of sample size calculation:

- Primary outcome variable – body weight change (%)
- Clinically significant effect = 5% (this value as clinically significant was presented in previous studies assessing the effect of drug therapy on body weight) [11].
- Standard deviation = 6.5 (this value obtained when processing the data of the pilot sample at the time of study initiation).
- Significance level = 5%
- Power = 80%
- Test – two-way

Sample size formula: $n = 2 \times (A+B)^2 / (d/SD)^2$, where n is the sample size for each group, SD is the standard deviation, d is the clinically significant effect, $A=1.96$ at the 5% significance level, $B=0.84$ at the 80% power level, $n = 2 \times (1.96+0.84)^2 / (5/6.5)^2$

In total, at least 26 subjects in each of the groups.

Methods of statistical analysis: statistical processing of the obtained data was carried out using the analytics software package STATISTICA (StatSoft Inc. USA, release 8.0). Quantitative data are presented as a median and interquartile range; qualitative data are presented as percentage values. For comparison between independent groups, a non-parametric method χ^2 with Yates correction was used for qualitative variables and the Mann-Whitney U-test for quantitative variables. Comparison of dependent groups by quantitative variables was performed using Wilcoxon test. Differences were considered statistically significant at $p < 0.05$. In multiple comparisons the significance criterion was recalculated using Bonferroni correction.

Table 1. Baseline characteristics of patients

Parameter	Group 1 (n=81)	Group 2 (n=75)	p
Age, years	40[36;44]	38[33;44]	0.144
Individual target weight (at BMI 24.9 kg/m ²), kg	80[76;84]	80[76;82]	0.397
Excess body weight relative to individual target, kg	31[20;38]	25[13;39]	0.104
Body weight, kg	110[100;118]	105[90;120]	0.181
BMI, kg/m ²	34.7[30.8;36.8]	32.3[28.9;36.6]	0.064
WC, cm	113[109;118]	112[102;122]	0.477
LH, IU/L	3.2[2.1;4.5]	2.9[2.3;4.1]	0.371
Testosterone, nmol/L	7.3[5.8;9.0]	7.6[6.1;8.8]	0.643
AMS score	38[33;42]	36[33;40]	0.114
Hematocrit, %	45,6[42,8;48,4]	44,9[42,3;47,7]	0.685
Hemoglobin, g/L	150[140;159]	148[141;158]	0.412

Note: Mann-Whitney U-test. Me[25%;75%] – quantitative data are presented in the form of medians and interquartile range boundaries. Bonferroni correction was applied, significance level $p < 0.0045$.

Table 2. Patient examination data over time

Parameter	Group 1 (n=81)	Group 2 (n=75)	p**
Weight – 6 months, kg	103[96;113]	99[86;114]	0.348
p (Body weight 0–6 months)	<0.001*	<0.001*	
Body weight 12 months, kg	98[93;108]	100[85;113]	0.970
p (Body weight 0–12 months)	<0.001*	<0.001*	
BMI – 6 months, kg/m ²	32.9[29.5;34.9]	30.1[27.4;34.6]	0.206
p (BMI 0–6 months)	<0.001*	<0.001*	
BMI – 12 months, kg/m ²	31.5[28.3;33.5]	30.0[27.3;34.4]	0.846
p (BMI 0–12 months)	<0.001*	<0.001*	
WC – 6 months, cm	109[102;113]	107[98;116]	0.645
p (WC 0–6 months)	<0.001*	<0.001*	
WC – 12 months, cm	104[97;108]	106[96;115]	0.087
p (WC 0–12 months)	<0.001*	<0.001*	
Testosterone – 6 months, nmol/L	16.7[14.6;19.8]	9.9[7.7;12.8]	<0.001
p (Testosterone 0–6 months)	<0.001*	<0.001*	
Testosterone – 12 months, nmol/L	16.4[14.3;18.3]	10.8[7.6;12.6]	<0.001
p (Testosterone 0–12 months)	<0.001*	<0.001*	
AMS – 6 months	28[26;31]	33[30;36]	<0.001
p (AMS 0–6 months)	<0.001*	<0.001*	
AMS – 12 months	27[25;29]	32[28;35]	<0.001
p (AMS 0–12 months)	<0.001*	<0.001*	
Hematocrit – 12 months, %	47.2[45.0;50.0]	45.1[43.0;47.5]	<0.001
p (Hematocrit – 0–12 months)	<0.001*	0.655	
Hemoglobin - 12 months, g/L	156[148;167]	148[141;155]	<0.001
p (Hemoglobin – 0–12 months)	<0.001*	0.727	

Note: *Wilcoxon test; **Mann-Whitney U test. Me[25%;75%] – quantitative data are presented in the form of medians and interquartile range boundaries. Since multiple comparisons were made, Bonferroni correction was applied, statistical significance level $p < 0.0042$. BMI, body mass index; WC, waist circumference; AMS, Androgen Deficiency Symptom Questionnaire score.

Table 3. Changes in studied parameters

Parameter	Group 1 (n=81)	Group 2 (n=75)	p
Δ of body weight loss from baseline 0–6 months, %	-4.8[-6.2;-3.8]	-2.9[-5.3;-1.4]	<0.001
Δ of body weight loss from baseline 0–12 months, %	-8.0[-10.4;-6.8]	-3.2[-7.5;-2.0]	<0.001
Δ of excess weight loss 0–6 months, %	-18.2[-24.9;-13.2]	-13.3[-30.7;-6.6]	<0.001
Δ of excess weight loss 0–12 months, %	-34.2[-47.6;-24.9]	-18.7[-34.6;-8.8]	<0.001
Δ BMI 0–6 months, kg/m ²	-1.7[-2.2;-1.1]	-1.1[-2.0;-0.5]	<0.001
Δ BMI 0–12 months, kg/m ²	-2.8[-3.6;-2.3]	-1.3[-2.6;-0.7]	<0.001
Δ WC 0–6 months, cm	-5[-7;-3]	-3[-6;-2]	0.050
Δ WC 0–12 months, cm	-9[-12;-7]	-4[-9;-3]	<0.001
Δ AMS score 0–6 months	-9[-12;-7]	-4[-7;-1]	<0.001
Δ AMS scores 0–12 months	-11[-13;-8]	-5[-8;-2]	<0.001

Note: Mann-Whitney U-test. Me[25%;75%] – quantitative data are presented in the form of medians and interquartile range boundaries. Since multiple comparisons were made, Bonferroni correction was applied, statistical significance level $p < 0.005$. BMI, body mass index; WC, waist circumference; AMS, Androgen Deficiency Symptom Questionnaire score.

Table 4. Results of retrospective analysis of hypogonadism outcomes

Parameter	Eugonadism (n=31)	Hypogonadism (n=44)	p
Age, years	36[33;43]	39[34;45]	0.076
LH, IU/L	2.9[2.4;4.2]	2.9[2.3;4.0]	0.953
Weight at baseline, kg	106[90;117]	105[91;129]	0.521
Weight – 6 months, kg	97[82;107]	103[89;126]	0.069
Δ of body weight loss from baseline 0–6 months, %	-6.0[-9.8;-4.0]	-2.0[-2.6;-1.0]	<0.0000
Body weight 12 months, kg	97[80;105]	102[88;127]	0.036
Δ of body weight loss from baseline 0–12 months, %	-8.5[-11.0;-5.9]	-2.3[-3.0;-1.6]	<0.0000
Individual target weight (at BMI 24.9 kg/m ²), kg	79[75;81]	80[76;84]	0.157
Excess weight relative to individual target at baseline, kg	24[14;36]	24[12;46]	0.501
Δ of excess weight loss 0–6 months, %	-28.3[-48.2;-16.4]	-8.8[-14.7;-4.6]	<0.0000
Δ of excess weight loss 0–12 months, %	-34.6[-61.2;-22.6]	-11.8[-18.0;-6.1]	<0.0000
BMI at baseline, kg/m ²	32.3[28.9;36.4]	31.9[28.8;37.5]	0.752
BMI – 6 months, kg/m ²	29.3[26.8;34.1]	30.7[28.5;36.8]	0.125
Δ BMI 0–6 months, kg/m ²	-2.0[-2.9;-1.4]	-0.6[-1.0;-0.3]	<0.0000
BMI – 12 months, kg/m ²	28.8[26.6;33.5]	30.8[28.3;36.6]	0.044
Δ BMI 0–12 months, kg/m ²	-2.9[-3.9;-2.1]	-0.9[-1.1;-0.6]	<0.0000
WC at baseline, cm	113[102;120]	111[102;124]	0.418
WC – 6 months, cm	104[96;111]	108[100;121]	0.015
Δ WC 0–6 months, cm	-7[-10;-5]	-2[-3;-1]	<0.0000
WC – 12 months, cm	104[93;109]	108[99;121]	0.003
Δ WC 0–12 months, cm	-9[-13;-7]	-3[-4;-2]	<0.0000
Testosterone, nmol/L	8.1[7.1;9.9]	7.3[5.9;8.4]	0.033
Testosterone – 6 months, nmol/L	13.0[12.1;13.8]	7.9[5.7;9.6]	<0.0000
Δ Testosterone 0–6 months, nmol/L	4.9[2.0;7.5]	0.5[-1.8;2.5]	<0.0000
Testosterone 12 months, nmol/L	12.7[12.3;13.9]	7.9[6.6;9.8]	<0.0000
Δ Testosterone 0–12 months, nmol/L	5.1[3.0;6.4]	1.0[-1.2;2.7]	<0.0000
AMS score at baseline	35[32;40]	36[34;42]	0.158
AMS score – 6 months	30[26;33]	35[32;38]	<0.0000
Δ AMS score 0–6 months	-6[-8;-4]	-2,0[-5;1]	<0.0000
AMS score – 12 months	28[25;31]	34[32;36]	<0.0000
Δ AMS scores 0–12 months	-7[-10;-5]	-3[-6;-1]	<0.0000
AMS Ps at baseline	9[8;11]	9[8;11]	0.987
AMS Ps – 12 months	8[7;8]	8[7;9]	0.049
AMS Som at baseline	15[13;17]	15[14;19]	0.437
AMS Som – 12 months	12[11;14]	14[12;16]	0.002
AMS Sex at baseline	11[10;13]	12[11;13]	0.006
AMS Sex – 12 months	9[8;10]	12[11;13]	<0.0000

Note: Mann-Whitney U-test; Me[25%;75%] – quantitative data are presented in the form of medians and interquartile range boundaries. Since multiple comparisons were made, Bonferroni correction was applied, statistical significance level $p < 0.0013$. LH, luteinizing hormone; BMI, body mass index; WC, waist circumference. AMS Ps, psychogenic symptoms, AMS Som, somatovegetative symptoms, AMS Sex, sexual symptoms.

RESULTS

Subjects (participants) of research

At the time of enrollment, the groups were comparable in terms of study parameters, see Table 1.

Primary study outcomes

Patient examination results over time are presented in Table 2.

In both groups 1 and 2, changes in most of the parameters studied were statistically significant, with the exception of hematocrit and hemoglobin levels in Group 2. When comparing the results achieved between the study groups, it was found that patients who received TRT at both 6 and 12 months from the date of inclusion in the study had a statistically significantly higher level of total testosterone and less severity of symptoms of androgenic deficiency (AMS scores). At the same time, hematocrit and hemoglobin levels in Group 1 were significantly higher than in Group 2 at the end of the study.

When assessing the change in the studied parameters after 12 months, it was found that patients receiving TRT achieved a statistically significant greater weight loss from baseline (mean weight change difference between groups -4.0% (95% CI -5.2 ; -2.7 , $p < 0.001$), greater excess weight loss, greater decrease in BMI and WC, and severity of androgen deficiency symptoms, see Table 3.

Secondary study outcomes

In the group with dietary and lifestyle corrections alone, 31 (41% (95% CI 30.1–53.3)) subjects achieved elimination of hypogonadism at 12 months from the start of the trial. Most of them (24 subjects, 32% (95% CI 21.7–43.8)) normalized the level of total testosterone within 6 months. The results of comparing the patient examination data depending on the outcome are presented in Table 4.

Patients with normalized testosterone production achieved a statistically significantly greater decrease in BMI, WC and the percentage of body weight loss from baseline (-8.5 [-11.0 ; -5.9], which is clinically significant), as well as excess weight loss compared to patients with persisting hypogonadism. At the same time, there were no differences in body weight, BMI and WC at baseline.

Adverse events

Hematocrit elevations above the reference value ($>50\%$) were observed in 23% of subjects on TRT and 12% of subjects without hypogonadism therapy, $p=0.098$ (χ^2 with Yates correction). Hemoglobin elevations above the reference value (>160 g/L) were detected in 37% and 15% of subjects on TRT and without hypogonadism therapy, respectively ($p=0.003$; χ^2 with Yates correction). There were no elevations in hematocrit and hemoglobin requiring TRT discontinuation.

DISCUSSION

Summary of the main result of the study

Patients who received TRT in combination with lifestyle and nutrition correction achieved a statistically significant greater weight loss (mean difference -4.0% (95% CI -5.2 ; -2.7 , $p < 0.001$)), a decrease in WC, as well

as in the severity of symptoms of androgen deficiency, compared patients with lifestyle and nutrition correction without androgen therapy. Elimination of hypogonadism with lifestyle and nutrition correction alone was achieved in 41% (95% CI 30.1–53.3) of patients with statistically significant and clinically significant weight loss, regardless of baseline BMI and WC.

Discussion of the primary study outcome

The data obtained in our study on the statistically significant positive effect of TRT of hypogonadism on metabolic and anthropometric indicators of obese men are consistent with a number of large studies and meta-analyses published recently [13, 14]. Assessment of the mechanisms of testosterone effect on metabolic parameters repeatedly demonstrated positive effect of testosterone on body composition with an increase in lean mass, lipolytic effect with a decrease in markers of aseptic inflammation, a decrease in leptin levels and an increase in adiponectin levels [15–17]. According to one of the largest studies of men with hypogonadism and obesity, TRT was associated with a significant decrease in fat content, an increase or maintaining of muscle mass, and a decrease in lipids, uric acid and WC [18]. These data are consistent with our findings of a statistically significantly greater reduction in WC with TRT compared to the control group, which reflects visceral fat loss. Another advantage of TRT was a statistically significant reduction in the symptoms of androgen deficiency, which was accompanied by an improvement in the quality of life not only in the sexual sphere, but also in the psycho-emotional and somatic statuses, and was consistent with the results published by other researches [14, 19]. At the same time, TRT was safe — we noted cases of increased hemoglobin and hematocrit, and these data correlate with the results of the TRAVERSE study, which did not reveal a significant relationship between the change in hematocrit and the risk of serious cardiovascular events when testosterone gel was used [20]. Thus, our results are consistent with the vast majority of data from other researchers and indicate the safety of TRT and its positive effect on the reduction of visceral fat mass.

STUDY LIMITATIONS

The sample was formed from patients who sought medical care from the large national medical center, therefore, the effectiveness of TRT in the general population of men with obesity and hypogonadism may differ. Given the association between weight loss and prospects for testosterone recovery, this factor will influence treatment outcome in other samples depending on its severity.

IMPRESSION

Treatment of hypogonadism using TRT has a positive effect on weight loss in obese males. Furthermore, normalization of testosterone levels leads to a reduction in androgen deficiency symptoms, accompanied by an improvement in quality of life (QoL). Elimination of hypogonadism with obesity treatment alone (without the use of TRT) is possible in 41% (95% CI 30.1–53.3) of cases, provided

clinically significant weight loss is achieved. Since most respondents achieved normalization of total testosterone levels within the first 6 months, this allows timely initiation of TRT to those patients who have not achieved stable normalization of total testosterone levels within a specified time.

ADDITIONAL INFORMATION

Source of funding. The study was carried out with the financial support from Besins Healthcare RUS.

Conflict of interest. R.V. Rozhivanov – received payments from Besins Healthcare RUS in 2017-2026 for conducting educational lectures, E.V. Morozova – no conflict of interest, V.A. Ioutsi – no conflict

of interest, L.V. Savelyeva – no conflict of interest, E.R. Rozhivanova – no conflict of interest, E.N. Andreeva – no conflict of interest, G.A. Melnichenko – received payments from Besins Healthcare RUS in 2017-2026 for conducting educational lectures, N.G. Mokrysheva – no conflict of interest.

Contribution of the authors. R.V. Rozhivanov – development of the study concept, collection and processing of research data, writing the text, E.V. Morozova – collecting research data, writing text, V.A. Ioutsi – laboratory research, M.A. Antsupova – laboratory research, L.V. Savelyeva – collecting research data, E.R. Rozhivanova – collecting literature, E.N. Andreeva – text editing, G.A. Melnichenko – text editing, N.G. Mokrysheva – text editing.

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