ISSN 2786-6661

Restoration of Blood Pressure Control Through Management of Obstructive Sleep Apnea and Optimization of Testosterone Therapy in a Patient with Hypergonadotropic Hypogonadism

UDC:616.1:612.4:616-03:616.6:615.2

DOI: https://doi.org/10.32345/USMYJ.1(160).2026.62-69

Received: January 06, 2026
Accepted: March 13, 2026
Published online: March 31, 2026

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Abstract: primary hypergonadotropic hypogonadism is an uncommon endocrine disorder resulting from primary testicular failure with preserved hypothalamic–pituitary regulation and requiring lifelong testosterone replacement therapy. In adult patients, this condition is frequently complicated by severe metabolic and cardiovascular disorders, particularly morbid obesity, arterial hypertension, and insulin resistance. The coexistence of these conditions significantly complicates both diagnostic evaluation and long-term therapeutic decision-making, especially when standard hormone replacement strategies fail to achieve stable biochemical and clinical control. To demonstrate the clinical significance of identifying and treating comorbid obstructive sleep apnea (OSA) syndrome as a prerequisite for effective testosterone replacement therapy and blood pressure control in a patient with hypergonadotropic hypogonadism and severe cardiometabolic comorbidities. A detailed clinical, laboratory, instrumental and follow-up assessment of a male patient with long-standing hypergonadotropic hypogonadism was performed, including endocrine, cardiological, and somnological evaluation. Therapeutic interventions and their outcomes were analyzed within a multidisciplinary management framework. We report the case of a 40-year-old male with a childhood diagnosis of primary hypergonadotropic hypogonadism who had been receiving long-term testosterone undecanoate therapy. Over time, the patient developed progressive morbid obesity (body mass index exceeding 50 kg/m^2) accompanied by metabolic disturbances, which prevented sustained normalization of serum testosterone levels despite ongoing treatment. He presented with recurrent severe headaches, dizziness, transient visual impairment, tremor, and two episodes of short-term loss of consciousness. Clinical examination revealed persistent arterial hypertension with blood pressure values reaching 170/105 mmHg, corresponding to grade II hypertension and very high cardiovascular risk. Conventional antihypertensive therapy failed to achieve adequate blood pressure control. Extended diagnostic evaluation identified previously undiagnosed OSA, recognized as an independent contributor to hypertension and a relative contraindication to intensification of testosterone therapy. Initiation of continuous positive airway pressure (CPAP) therapy resulted in marked clinical improvement and enabled safe reassessment of testosterone replacement. Subsequent reduction of testosterone undecanoate injection intervals combined with adjunctive aromatase inhibitor therapy led to stable normalization of serum testosterone concentrations. This hormonal correction was associated with sustained blood pressure control below 130/85 mmHg without escalation of antihypertensive treatment and

How to cite this article: Shkvarok-Lisovenko A, Korost Y. Restoration of blood pressure control through management of obstructive sleep apnea and optimization of testosterone therapy in a patient with hypergonadotropic hypogonadism: a case report. Ukrainian Scientific Medical Youth Journal. 2026;1(160):62-69. doi:10.32345/USMYJ.1(160).2026.62-69

complete resolution of neurological symptoms. The achieved clinical stability persisted throughout follow-up. This clinical case highlights the complex and often underestimated interplay between hypergonadotropic hypogonadism, morbid obesity, OSA and arterial hypertension. It underscores the importance of a stepwise, pathophysiology-driven diagnostic approach, in which recognition and treatment of sleep-disordered breathing represent a critical prerequisite for the safe and effective optimization of testosterone replacement therapy. The case further emphasizes the necessity of a coordinated multidisciplinary strategy involving family physicians, endocrinologists, cardiologists, somnologists and andrologists to achieve sustainable clinical outcomes in patients with combined endocrine and cardiovascular comorbidities.

Key words: Hypergonadotropic Hypogonadism, Hypertension, Obesity, Obstructive Sleep Apnea

Introduction

Hypergonadotropic hypogonadism (HH) is a pathological condition characterized by impaired gonadal function in the presence of preserved activity of the hypothalamic-pituitary axis; in primary male hypogonadism, testicular dysfunction leads to insufficient testosterone production and compensatory pituitary hyperstimulation, while clinical manifestations include infertility, delayed or impaired sexual development, decreased libido, and other systemic symptoms [1, 2]. Reduced testosterone levels are closely associated with obesity, which is frequently observed in patients with HH; in men with a body mass index exceeding 35–40 kg/m², total and free testosterone levels are reduced by more than 50% compared with lean individuals, and testosterone deficiency itself is associated with decreased libido, sexual dysfunction, fatigue, reduced physical strength, depressive symptoms, and cognitive impairment, despite the relative rarity of this condition and the insufficient investigation of its metabolic and cardiovascular consequences [3, 4]. Accumulating evidence suggests that testosterone replacement therapy, including testosterone undecanoate, may contribute to improved blood pressure control in patients with HH and arterial hypertension, and that long-term testosterone therapy improves hemodynamic parameters, likely through enhanced vascular elasticity, reduced insulin resistance, attenuation of visceral adiposity, and suppression of systemic inflammation – key mechanisms in the pathogenesis of arterial hypertension [5, 6, 7].

Aim

To demonstrate the importance of a stepwise, multidisciplinary approach to restoring blood pressure control in a patient with hypergonadotropic hypogonadism by optimizing testosterone replacement therapy following the diagnosis and treatment of OSA through a clinical case.

Materials and methods

A detailed clinical case analysis was performed, including longitudinal assessment of hormonal, metabolic, cardiovascular, and instrumental parameters in a patient with hypergonadotropic hypogonadism, arterial hypertension, obesity, and OSA during staged therapeutic interventions. Overnight cardiorespiratory monitoring confirmed OSA.

Case Presentation

A 40-year-old man from Ukraine who had been diagnosed in childhood with hypergonadotropic hypogonadism sought care from his family physician because of recurring headaches. These headaches were accompanied by dizziness, disturbances of the visual fields, memory difficulties and tremor of the left hand with sensory complaints. The neurological symptoms were most noticeable when the headache reached its maximum intensity. Over the previous month the patient had also experienced two brief episodes of loss of consciousness. He reported no history of smoking, alcohol use or illicit drug consumption. During the two months leading up to the consultation headache episodes occurred as often as four times per week and the patient subjectively linked their onset to abrupt weather changes during the summer period. For several years he had been on continuous testosterone replacement therapy administered as intramuscular testosterone undecanoate (1000 mg/4 mL). The treatment regimen began with two injections given one month apart followed by maintenance injections every 12 weeks. Klinefelter syndrome had previously been ruled out as a potential cause of his condition. The most recent hormonal assessment was performed in June 2025 (Table 1), approximately six weeks before presentation and coincided with his scheduled testosterone injection. At the start of testosterone therapy in 2020 the patient's body weight was 152 kg with a total testosterone level of 4.86 nmol/L and a leptin concentration of 13.69 ng/mL. Routine instrumental evaluations were carried out in line with andrological guidelines including a scrotal ultrasound performed in July 2025 (Figure 1). Ultrasonography demonstrated markedly reduced testicular volumes while intratesticular blood flow remained preserved. Both testes showed a heterogeneous echotexture caused by numerous small hyperechoic foci consistent with testicular microlithiasis. No focal

ISSN 2786-6661

Table 1. Laboratory parameters of the patient in June 2025

Diagnostic Caption	Result	Reference Range
Luteinizing hormone (mIU/mL)	10.8	0.8-7.6
Thyroid-stimulating hormone (μIU/mL)	0.81	0.4-4.0
Follicle-stimulating hormone (mIU/mL)	25.0	0.7-11.1
Prolactin (μIU/mL)	8.10	1.9-25.0
Estradiol (pg/mL)	20	7.63-56.0
Leptin (ng/mL)	16.46	2.05-5.63
Total testosterone (nmol/L)	13.86	15-50

lesions, hydrocele or varicocele were detected. Color Doppler assessment revealed no venous reflux during the Valsalva maneuver.

At the time of presentation in August 2025 the patient was found to have persistently elevated blood pressure measuring 160/100 mmHg. Heart rate was 80 beats per minute and peripheral oxygen saturation remained within normal limits. His body weight had increased to 181 kg at a height of 187 cm corresponding to a BMI of 51.8 kg/m². During the visit he received a single oral dose of captopril 25 mg in combination with hydrochlorothiazide 12.5 mg which led to a decrease in blood pressure to 130/80 mmHg by the end of the consultation. The

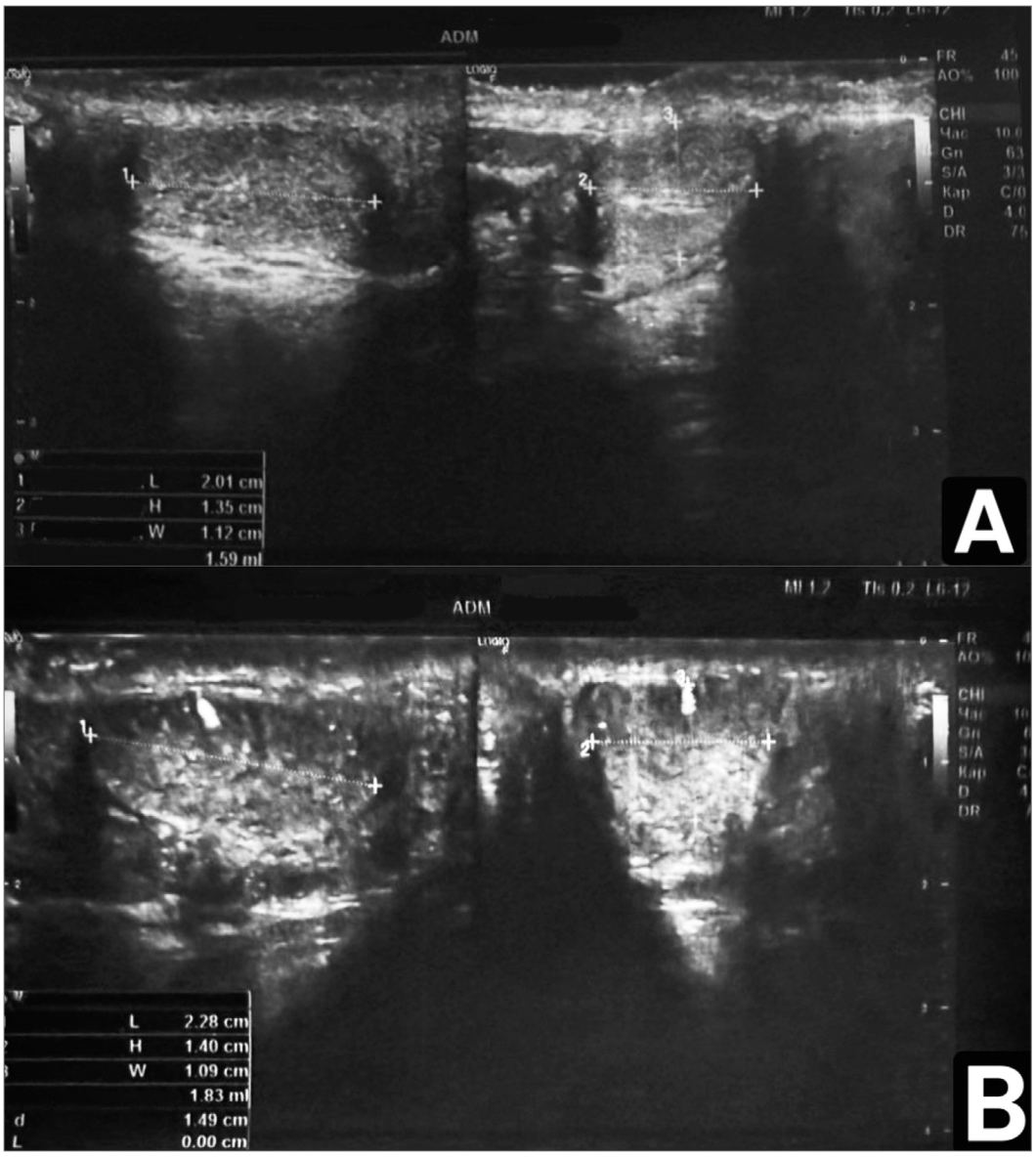

Figure 1: Scrotal ultrasound demonstrating bilateral testicular hypoplasia and testicular microlithiasis. A. Right testis showing markedly reduced volume with heterogeneous echotexture due to multiple punctate hyperechoic foci consistent with testicular microlithiasis and preserved intratesticular blood flow. B. Left testis demonstrating similar findings, including reduced volume, heterogeneous echotexture with punctate hyperechoic foci and preserved vascularity.

involuntary movements of the hand noted previously resolved following blood pressure normalization. To rule out secondary neurological causes including focal epilepsy or intracranial tumors the patient was referred for inpatient neurological assessment. In September 2025 he was admitted to the neurology department. MRI of the brain and spinal cord was planned but technical constraints related to excessive body weight allowed imaging of the brain and cervical spine only (Figure 2). MRI findings included multiple small areas of altered signal intensity within the cerebral white matter consistent with cerebral microangiopathy (Fazekas grade I-II). No signs of intracranial neoplasms, acute ischemic changes, hemorrhage or demyelinating disease were identified and the McDonald 2024 criteria for multiple sclerosis were not fulfilled [8].

Given repeated blood pressure elevations up to 170/105 mmHg a cardiology consultation was obtained. Comprehensive laboratory testing was performed including lipid profile, glucose, glycated hemoglobin, testosterone, adrenocorticotropic hormone and vitamin D levels (Table 2). Based on the overall findings the patient was diagnosed with grade II arterial hypertension, stage 2, associated with a very high cardiovascular risk. Overnight cardiorespiratory monitoring confirmed OSA with a clinically significant frequency of respiratory events (apnea-hypopnea index (AHI) >15 events/hour), corresponding to at least moderate OSA. Intermittent oxygen desaturations were recorded, with nadir oxygen saturation below 90%, consistent with clinically relevant nocturnal hypoxemia. Vitamin D deficiency was also identified.

After hospital discharge the family physician formulated a comprehensive and stepwise management plan. The diagnostic and therapeutic approach began with consultation by a somnologist and assessment for continuous positive airway pressure (CPAP) therapy since untreated OSA is

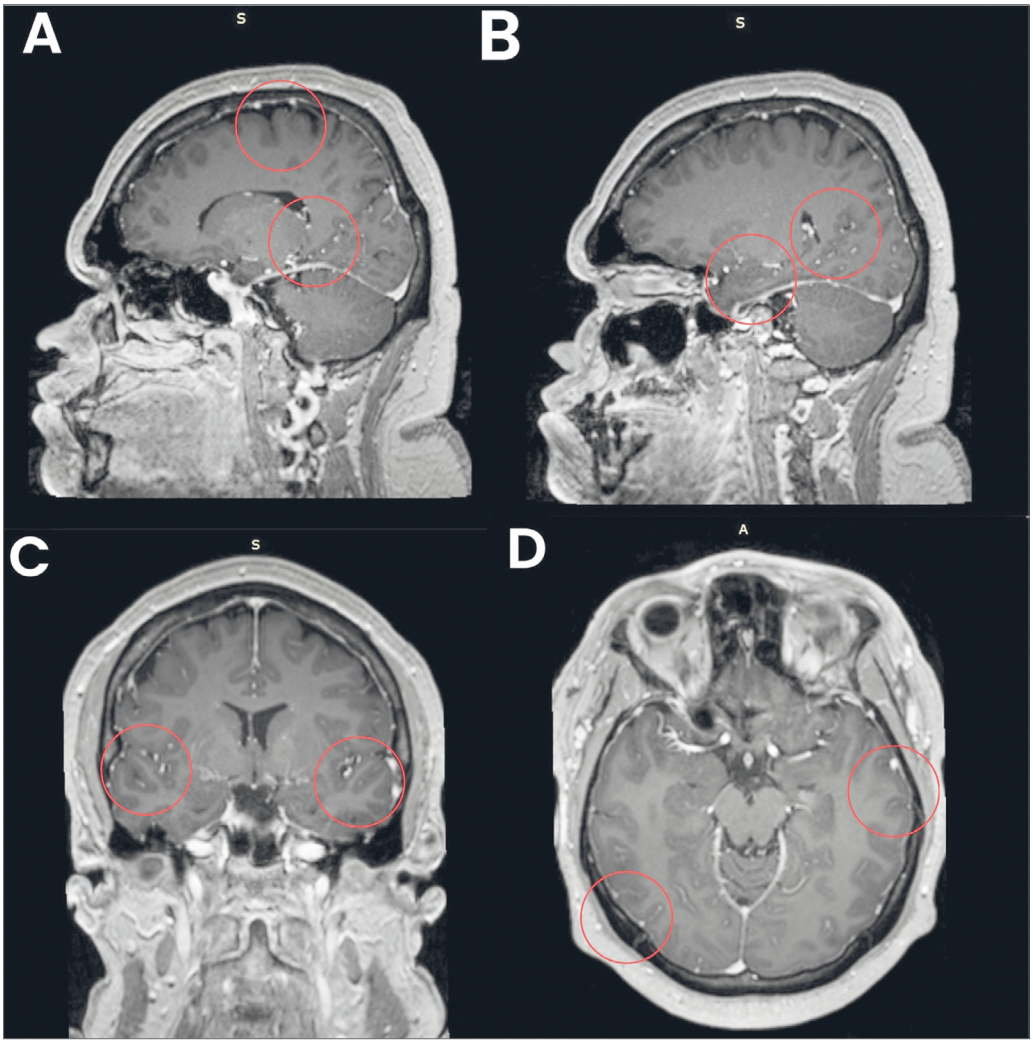

Figure 2: Brain magnetic resonance imaging demonstrating cerebral white matter changes of vascular origin. Sagittal (A, B), frontal (C), and axial (D) MRI sequences show multiple small hyperintense foci within the cerebral white matter (red circles), consistent with chronic microangiopathic changes (Fazekas grade I-II) [8].

ISSN 2786-6661

Table 2: Laboratory parameters during hospitalization in September 2025

Diagnostic Caption	Result	Reference Range
Total cholesterol (mmol/L)	4.5	< 5.0
Low-density lipoprotein cholesterol (mmol/L)	2.8	< 3.0
High-density lipoprotein cholesterol (mmol/L)	1.0	> 1.0
Triglycerides (mmol/L)	1.6	< 1.7
Atherogenic index	3.5	2-3
Glucose (mmol/L)	6.1	4.0-5.5
Adrenocorticotropic hormone (pg/mL)	22.5	6-58
Total testosterone (nmol/L)	13.3	15-50
Total vitamin D (ng/mL)	18.66	30-70

considered a relative contraindication to testosterone replacement therapy according to clinical guidelines, due to the potential risk of worsening respiratory events and erythrocytosis. Correction of this sleep-related breathing disorder was therefore prioritized as an essential prerequisite before addressing testosterone deficiency which in turn was necessary for effective blood pressure control. Antihypertensive treatment was initiated with azilsartan medoxomil 40 mg and felodipine 2.5 mg administered once daily along with vitamin D3 supplementation at a dose of 5600 IU per day for three months. The patient was referred for further evaluation by a somnologist and an andrologist. Management of OSA remained the primary focus. CPAP therapy was delivered using an auto-adjusting device with pressure range applied in moderate-to-severe OSA (approximately 8-14 cm H_2O). Clinical follow-up indicated satisfactory adherence (≥4 hours per night on most nights).

Following improvement in sleep quality and stabilization of respiratory parameters reassessment of testosterone replacement therapy was undertaken. Because testosterone levels remained subtherapeutic with the prior dosing schedule the injection interval for testosterone undecanoate was shortened to nine weeks. In addition anastrozole 0.5 mg twice weekly was introduced. Over the subsequent three months total testosterone concentrations increased to 17 nmol/L and blood pressure stabilized below 130/85 mmHg without the need for further adjustments to antihypertensive treatment. Headaches and tremor resolved completely and no recurrence of symptoms was reported through January 2026. During a four-month follow-up period after initiation of CPAP therapy and adjustment of testosterone replacement, clinical and hemodynamic parameters remained stable.

Discussion

This case illustrates the complex interplay between hypergonadotropic hypogonadism, morbid obesity, arterial hypertension, and OSA. Longstanding primary hypogonadism combined with progressive obesity likely contributed to persistent testosterone deficiency despite ongoing replacement therapy. Obesity exacerbates hypogonadism through increased aromatization of androgens, elevated leptin levels, and insulin resistance, further suppressing endogenous testosterone production [3, 4]. The sequence in which interventions were applied in this case was not arbitrary: it was dictated by the pathophysiology of the conditions and their reciprocal interactions.

First, consultation with a somnologist and addressing the potential need for CPAP therapy was essential. Untreated OSA is not only an independent risk factor for hypertension and cardiovascular morbidity, but also represents a relative contraindication to testosterone replacement therapy (TRT) according to clinical guidelines, due to potential worsening of respiratory events and adverse cardiovascular effects if initiated in the setting of unaddressed sleep-disordered breathing [9]. Without proper management of OSA, initiating testosterone undecanoate could have exacerbated hypoxia, increased hematocrit, and worsened hypertension, undermining both hormonal and cardiovascular outcomes. The absence of ambulatory blood pressure monitoring represents a limitation of this case report and does not allow objective evaluation of nocturnal blood pressure patterns. Although erythrocytosis is a recognized complication of testosterone replacement therapy, hematocrit levels were monitored during follow-up and remained within the reference range throughout treatment optimization. No laboratory evidence of polycythemia was observed in this patient.

Indeed, OSA contributes to a cycle of hormonal dysregulation by disrupting sleep architecture, increasing sympathetic activity, and promoting intermittent hypoxia – all of which impair hypothalamic-pituitary-gonadal axis function and may worsen hypogonadism [9]. This physiological interplay is supported by observational evidence linking OSA severity with reduced testosterone levels in men with obesity. For example, clinical studies have shown that individuals with OSA and obesity often have significantly lower serum testosterone, and that the coexistence of these conditions correlates with

more profound endocrine dysfunction compared to obesity alone [10].

Cases in the literature where this sequence was not observed often report suboptimal or adverse outcomes. Patients initiated on TRT without prior sleep apnea assessment have exhibited exacerbated hypertension, polycythemia, or worsening respiratory function, underscoring the danger of overlooking OSA in the diagnostic workup [9]. These reports reinforce the necessity of screening for and managing sleep-disordered breathing before hormonal interventions in patients with complex metabolic profiles.

Testosterone deficiency has been linked to endothelial dysfunction, increased arterial stiffness, and heightened sympathetic nervous system activity, all of which contribute to the development and persistence of arterial hypertension [1, 2, 11]. OSA represents an independent risk factor for hypertension and a relative contraindication to testosterone therapy when untreated, underscoring the importance of timely diagnosis and management [4, 12]. Only after initiation of CPAP therapy, optimization of testosterone dosing, and stabilization of antihypertensive treatment were target hormonal and blood pressure values achieved. These findings are consistent with previously published clinical evidence supporting a multidisciplinary approach to managing complex endocrine and cardiovascular comorbidities [5, 6]. Although improvement in serum testosterone levels coincided with stabilization of blood pressure, it is likely that adequate control of obstructive sleep apnea played a central pathophysiological role in restoring blood pressure regulation. Hormonal optimization may have contributed additionally through metabolic and vascular mechanisms; however, a direct causal relationship cannot be definitively established in a single-case observation.

Conclusions

This case underscores that the timely diagnosis and treatment of OSA is not a peripheral consideration but a central component of the therapeutic algorithm in patients with overlapping endocrine and cardiovascular pathologies. Only after stabilizing respiratory function with CPAP can clinicians safely pursue testosterone replacement - facilitating not only normalization of hormonal levels but also more effective control of blood pressure and reduction of long-term cardiovascular risk. Such a sequence aligns with emerging evidence advocating a personalized, stepwise strategy that addresses the most impactful and potentially modifiable risk factors first.

Funding. None.

Conflict of interest. The authors declare no conflict of interest.

Consent for publication. All authors of the article are acquainted with the final version of the manuscript and have no objections to its publication.

Patient Consent. Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Ethics Approval Statement. The case report was reviewed and approved by the Commission on Bioethics and Research Ethics of Bogomolets National Medical University, Kyiv, Ukraine, Protocol №203, 23.02.2026.

AI Statement. The authors confirm that no generative artificial intelligence tools were used in the preparation of this manuscript.

Author Contributions (CRediT). Conceptualization – Anastasiia Shkvarok-Lisovenko; Yaroslava Korost. Methodology – Anastasiia Shkvarok-Lisovenko; Yaroslava Korost. Software – Anastasiia Shkvarok-Lisovenko. Validation – Anastasiia Shkvarok-Lisovenko; Yaroslava Korost. Formal Analysis – Anastasiia Shkvarok-Lisovenko; Yaroslava Korost. Investigation – Anastasiia Shkvarok-Lisovenko. Resources – Anastasiia Shkvarok-Lisovenko. Data Curation – Anastasiia Shkvarok-Lisovenko; Yaroslava Korost. Writing – Original Draft Preparation – Anastasiia Shkvarok-Lisovenko. Writing – Review & Editing – Yaroslava Korost. Visualization – Anastasiia Shkvarok-Lisovenko. Supervision – Yaroslava Korost. Project Administration – Anastasiia Shkvarok-Lisovenko; Yaroslava Korost

ISSN 2786-6661

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Відновлення контролю артеріального тиску шляхом лікування обструктивного апное сну та оптимізації терапії тестостероном у пацієнта з гіпергонадотропним гіпогонадизмом

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Анотація. Первинний гіпергонадотропний гіпогонадизм є рідкісним ендокринним захворюванням, що характеризується первинною недостатністю функції яєчок за збереженої активності гіпоталамо-гіпофізарної осі та потребує довічної замісної терапії тестостероном. Незважаючи на відносно низьку поширеність, це захворювання часто поєднується з тяжкими метаболічними та серцево-судинними коморбідними станами, зокрема морбідним ожирінням, артеріальною гіпертензією, інсулінорезистентністю та порушеннями дихання під час сну, що значно ускладнює клінічне ведення таких пацієнтів і вимагає індивідуалізованого мультидисциплінарного підходу. У цій роботі представлено клінічний випадок 40-річного пацієнта з дитячим анамнезом гіпергонадотропного гіпогонадизму, який протягом кількох років отримував замісну терапію тестостерону ундеканоатом, однак на тлі прогресуючого морбідного ожиріння (ІМТ > 50 кг/м^2) та супутніх метаболічних порушень не вдавалося досягти стійкої нормалізації рівня тестостерону. Пацієнт звернувся по медичну допомогу зі скаргами на рецидивні інтенсивні головні болі, епізоди запаморочення, транзиторні порушення зору, тремор та два епізоди короткочасної втрати свідомості. Під час обстеження було виявлено стійке підвищення артеріального тиску до 170/105 мм рт. ст., що відповідало артеріальній гіпертензії II ступеня з дуже високим серцево-судинним ризиком. Незважаючи на проведення антигіпертензивної терапії, адекватного контролю артеріального тиску не вдавалося досягти до моменту корекції ендокринних порушень та порушень сну. У процесі обстеження було діагностовано синдром обструктивного апное сну, який є незалежним фактором ризику артеріальної гіпертензії та відносним протипоказанням до оптимізації замісної терапії тестостероном. Виявлення та лікування обструктивного апное сну із застосуванням CPAP-терапії стало ключовим етапом ведення пацієнта, оскільки дозволило безпечно переглянути режим введення тестостерону ундеканоату та досягти цільових концентрацій гормону в сироватці крові. Після корекції дихальних порушень під час сну, зменшення інтервалів між ін'єкціями тестостерону та додаткової терапії інгібітором ароматази було зафіксовано нормалізацію рівня тестостерону, стабілізацію артеріального тиску нижче 130/85 мм рт. ст. без потреби в ескалації антигіпертензивної терапії, а також повне зникнення неврологічної симптоматики. Досягнутий клінічний ефект зберігався протягом подальшого спостереження. Представлений клінічний випадок демонструє складну патофізіологічну взаємодію між гіпергонадотропним гіпогонадизмом, морбідним ожирінням, синдромом обструктивного апное сну та артеріальною гіпертензією. Він підкреслює принципову важливість поетапної діагностики та лікування, при якій усунення порушень дихання під час сну є необхідною передумовою для безпечної та ефективної оптимізації замісної терапії тестостероном і досягнення контролю артеріального тиску. Цей випадок підтверджує доцільність мультидисциплінарного підходу за участі сімейного лікаря, ендокринолога, кардіолога, сомнолога та андролога у веденні пацієнтів із поєднаними ендокринними та серцево-судинними захворюваннями.

Ключові слова: гіпергонадотропний гіпогонадизм, артеріальна гіпертензія, ожиріння, обструктивне апное сну.