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FUNCTIONAL STATE OF THE SYMPATHOADRENAL SYSTEM IN PATIENTS DIAGNOSED WITH ESSENTIAL HYPERTENSION

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Abstract

Background: Essential hypertension is a widespread cardiovascular disorder. The hyperactivation of the sympathoadrenal system (SAS) is considered one of the primary pathogenetic mechanisms leading to the elevation of blood pressure and subsequent target organ damage. Objective: To evaluate the functional state of the sympathoadrenal system by assessing catecholamine levels in patients diagnosed with essential hypertension. Methods: The study included 78 patients with Stage II essential hypertension and a control group of 20 healthy individuals. The functional state of the SAS was evaluated by measuring plasma levels of epinephrine (adrenaline) and norepinephrine (noradrenaline) using high-performance liquid chromatography. Hemodynamic parameters, including systolic and diastolic blood pressure, and heart rate, were also recorded. Results: Patients with hypertension exhibited a significant increase in SAS activity. Plasma norepinephrine levels were significantly higher in the hypertensive group (412.5 ± 35.2 pg/mL) compared to the control group (265.4 ± 22.8 pg/mL, $p < 0.05$), indicating elevated sympathetic nervous system tone. Epinephrine levels were also increased (74.3 ± 8.1 pg/mL vs. 42.1 ± 5.4 pg/mL, $p < 0.05$). A strong positive correlation was found between plasma norepinephrine levels and both systolic blood pressure and heart rate. Conclusion: Essential hypertension is characterized by marked hyperactivation of the sympathoadrenal system. This sustained sympathetic overdrive contributes to the maintenance of high blood pressure and highlights the necessity of incorporating sympatholytic or neuromodulatory agents in the comprehensive management of these patients.

Keywords

essential hypertension, sympathoadrenal system, catecholamines, norepinephrine, epinephrine, sympathetic nervous system.

ФУНКЦИОНАЛЬНОЕ СОСТОЯНИЕ СИМПАТОАДРЕНАЛОВОЙ СИСТЕМЫ У ПАЦИЕНТОВ С УСТАНОВЛЕННОЙ ГИПЕРТОНИЕЙ

Аннотация

Обоснование: Гипертоническая болезнь является распространенным сердечно-сосудистым заболеванием. Гиперактивация симпатоадреналовой системы (САС) считается одним из основных патогенетических механизмов, приводящих к повышению артериального давления и последующему поражению органов-мишеней. Цель исследования: Оценить функциональное состояние симпатоадреналовой системы путем определения уровня катехоламинов у пациентов с диагностированной гипертонической болезнью. Методы: В исследование было включено 78 пациентов с гипертонической болезнью II стадии и контрольная группа из 20 здоровых лиц. Функциональное состояние САС оценивали путем измерения уровня адреналина и норадреналина в плазме крови методом высокоэффективной жидкостной хроматографии. Также регистрировались гемодинамические показатели (САД, ДАД, ЧСС). Результаты: У пациентов с гипертонией

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выявлено значительное повышение активности САС. Уровень норадреналина в плазме крови был достоверно выше в группе гипертоников (412,5 ± 35,2 пг/мл) по сравнению с контрольной группой (265,4 ± 22,8 пг/мл, p < 0,05). Уровни адреналина также были повышены (74,3 ± 8,1 пг/мл против 42,1 ± 5,4 пг/мл, p < 0,05). Обнаружена сильная положительная корреляция между уровнем норадреналина и систолическим артериальным давлением. Заключение: Гипертоническая болезнь характеризуется выраженной гиперактивацией симпатоадреналовой системы. Это стойкое симпатическое перенапряжение способствует поддержанию высокого артериального давления.

Ключевые слова

гипертоническая болезнь, симпатоадреналовая система, катехоламины, норадреналин, адреналин.

GIPERTONIYA KASALĺIGI ANIQLANGAN BEMORLARDA SIMPATO ADRENAL TIZIMNI FUNKSIONAL HOLATI

Annotatsiya

Dolzarbligi: Gipertoniya kasalligi (GK) yurak-qon tomir kasalliklari orasida eng keng tarqalgan patologiyalardan biri hisoblanadi. Simpato-adrenal tizimning (SAT) giperaktivatsiyasi qon bosimining ko'tarilishi va nishon-a'zolar shikastlanishiga olib keluvchi asosiy patogenetik mexanizmlardan biri sifatida qaraladi. Maqsad: Gipertoniya kasalligi aniqlangan bemorlarda katexolaminlar miqdorini aniqlash orqali simpato-adrenal tizimning funksional holatini baholash. Material va metodlar: Tadqiqotga II bosqich gipertoniya kasalligi bilan og'rigan 78 nafar bemor va 20 nafar sog'lom shaxslardan iborat nazorat guruhi jalb etildi. SAT funksional holati qon plazmasidagi adrenalin va noradrenalin miqdorini yuqori samarali suyuqlik xromatografiyasi usulida aniqlash orqali baholandi. Natijalar: Gipertoniya bilan kasallangan bemorlarda SAT faolligining sezilarli darajada oshishi kuzatildi. Qon plazmasidagi noradrenalin miqdori gipertoniya guruhida (412,5 ± 35,2 pg/ml) nazorat guruhiga (265,4 ± 22,8 pg/ml, p < 0,05) nisbatan ishonchli darajada yuqori bo'ldi. Adrenalin miqdori ham yuqori ekanligi aniqlandi. Xulosa: Gipertoniya kasalligi simpato-adrenal tizimning yaqqol giperaktivatsiyasi bilan kechadi. Ushbu davomiy simpatik zo'riqish yuqori qon bosimini saqlab turishga xizmat qiladi.

Kalit so'zlar

gipertoniya kasalligi, simpato-adrenal tizim, katexolaminlar, noradrenalin, adrenalin.

INTRODUCTION

Essential hypertension (EH) remains a cornerstone issue in contemporary cardiology, functioning as a primary risk factor for catastrophic cardiovascular events such as myocardial infarction, ischemic stroke, and progressive heart failure. According to current epidemiological data, abnormal blood pressure elevation affects nearly a third of the global adult population, imposing a massive burden on healthcare systems [1].

The regulation of arterial blood pressure is a highly complex process mediated by interacting neurohumoral mechanisms. Among these, the sympathoadrenal system (SAS) and the renin-angiotensin-aldosterone system (RAAS) are the primary drivers [2]. A growing body of clinical evidence indicates that chronic hyperactivation of the sympathetic nervous system does not merely trigger transient spikes in blood pressure; rather, it acts as a fundamental catalyst for long-term structural remodeling within the vasculature and myocardial tissue [3].

Evaluating the functional status of the SAS typically involves quantifying circulating catecholamines—specifically norepinephrine and epinephrine—in the blood or urine.

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Norepinephrine, primarily released from sympathetic nerve terminals, directly increases total peripheral vascular resistance (TPVR) via alpha-1 adrenergic receptor stimulation. Conversely, epinephrine, secreted largely by the adrenal medulla, predominantly influences cardiac output by enhancing the chronotropic and inotropic properties of the heart through beta-1 receptor activation [4, 5].

Particularly during the early and moderate stages of EH, sympathoadrenal overactivity serves as an initiating pathogenic trigger, a concept heavily supported by international researchers [6, 7]. However, the exact nuances of catecholamine metabolism and their specific correlations with hemodynamic parameters in the local demographic—specifically among patients in the Andijan region—have not been comprehensively detailed in recent literature.

Therefore, the primary objective of this study was to evaluate the functional state of the sympathoadrenal system by analyzing plasma catecholamine levels in patients with essential hypertension and to determine how these neurohumoral shifts impact the clinical and hemodynamic course of the disease.

MATERIALS AND METHODS

Study Population - The clinical study was conducted at the Department of Faculty Therapy, based in the clinic of the Andijan State Medical Institute. We enrolled 78 patients diagnosed with Stage II essential hypertension, in accordance with the European Society of Cardiology (ESC) guidelines. This main study group (Group 1) consisted of 42 men and 36 women, with a mean age of 54.3 ± 6.8 years.

To establish baseline comparative data, a Control Group of 20 healthy volunteers, matched for age and gender without any history of cardiovascular or endocrine pathology, was concurrently examined.

Patients with secondary (symptomatic) hypertension, a history of acute myocardial infarction or stroke within the past 6 months, severe congestive heart failure (NYHA Class III-IV), uncontrolled diabetes mellitus, chronic kidney disease, or thyroid dysfunction were explicitly excluded from the study cohort. All participants provided informed consent prior to clinical evaluation.

Laboratory Procedures and Examinations - Comprehensive clinical assessments were performed for all subjects, encompassing detailed medical history gathering, physical examination, electrocardiography (ECG), and echocardiography. Arterial blood pressure was measured utilizing the standard Korotkov technique.

Catecholamine Assessment: To accurately gauge the sympathoadrenal state and minimize stress-induced hormone fluctuations, venous blood sampling was strictly scheduled between 08:00 and 09:00 AM. Patients were required to rest in a supine position for at least 20 minutes prior to venipuncture. Plasma concentrations of norepinephrine and epinephrine were quantified using high-performance liquid chromatography (HPLC) coupled with an electrochemical detector (Agilent Technologies, USA) utilizing standardized reagent kits. Values were recorded in picograms per milliliter (pg/mL).

Statistical Analysis - All acquired data were processed using SPSS Statistics, version 26.0 (IBM Corp., USA). The Shapiro-Wilk test was applied to verify the normal distribution of quantitative variables. Results are presented as the arithmetic mean alongside the standard deviation (M ± SD). Statistical significance of differences between the groups was determined via Student's t-test for independent samples, with a p-value of < 0.05 considered statistically significant. Pearson's correlation coefficient (r) was utilized to assess the strength of linear relationships between evaluated parameters.

AMERICAN ACADEMIC PUBLISHER
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RESULTS

Initial analysis of clinical and hemodynamic characteristics confirmed that blood pressure parameters in patients with essential hypertension were significantly higher than those in the control group (Table 1). Concurrently, a statistically significant elevation in heart rate (HR) was observed in the EH group. This resting tachycardia serves as an indirect yet reliable clinical indicator of heightened sympathetic nervous system tone.

Table 1. Clinical and hemodynamic characteristics of the examined groups (M ± SD)

Parameter	Control Group (n=20)	Main Group (EH, n=78)
Age (years)	52.4 ± 5.5	54.3 ± 6.8
Body Mass Index (kg/m²)	24.6 ± 2.1	28.9 ± 3.4*
Systolic BP (mmHg)	118.5 ± 6.2	164.2 ± 11.5*
Diastolic BP (mmHg)	76.4 ± 5.1	102.8 ± 7.4*
Heart Rate (bpm)	72.5 ± 4.3	86.4 ± 6.5*

*Note: * denotes $p < 0.05$ compared to the Control Group.*

Direct measurement of circulating plasma catecholamines provided definitive evidence of sympathoadrenal hyperactivation in the hypertensive cohort (Table 2). Specifically, the level of norepinephrine—the primary neurotransmitter of the sympathetic nervous system—was approximately 55% higher in the EH group compared to healthy controls ($p < 0.05$).

Similarly, the hormonal limb of the SAS, represented by epinephrine levels, demonstrated a pronounced increase of nearly 76% in patients with hypertension, reflecting a systemic neurohumoral stress response.

Table 2. Plasma catecholamine levels in the study population (M ± SD)

Parameter	Control Group (n=20)	Main Group (EH, n=78)
Norepinephrine (pg/mL)	265.4 ± 22.8	412.5 ± 35.2*
Epinephrine (pg/mL)	42.1 ± 5.4	74.3 ± 8.1*
Norepinephrine/Epinephrine Ratio	6.3 ± 0.8	5.5 ± 0.7

*Note: * denotes $p < 0.05$ compared to the Control Group.*

Further statistical evaluation using correlation analysis uncovered a direct pathogenetic link between SAS overactivity and hemodynamic disturbances. In the patient group, a strong positive correlation was identified between plasma norepinephrine levels and systolic blood pressure ($r = 0.68$; $p < 0.01$). Additionally, a significant positive correlation was found between circulating norepinephrine and resting heart rate ($r = 0.55$; $p < 0.05$). These data robustly support the concept that elevated catecholamines are directly responsible for the vascular spasm and chronotropic changes seen in these patients.

DISCUSSION

The results obtained from our investigation clearly demonstrate that sympathoadrenal dysfunction is not a mere secondary phenomenon but a pivotal factor in the pathogenesis of essential hypertension. The markedly elevated levels of both norepinephrine and epinephrine

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
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documented in our cohort align closely with findings reported in contemporary international literature [8, 9].

Physiologically, a persistent state of SAS activation and the continuous release of excessive norepinephrine into the bloodstream leads to the intense stimulation of alpha-1 adrenergic receptors located in the peripheral vascular bed. This sustained vasoconstriction invariably increases total peripheral vascular resistance, directly translating into elevated systemic blood pressure [10]. Concurrently, the elevated epinephrine levels act upon beta-1 adrenergic receptors, increasing both the force and rate of myocardial contractions. This is clinically mirrored in our study by the elevated resting heart rate of 86.4 ± 6.5 bpm observed in the hypertensive group.

Beyond acute hemodynamic shifts, chronic sympathetic overactivity exerts profound trophic (structural) effects. Norepinephrine acts as a potent mitogen for vascular smooth muscle cells and cardiomyocytes, promoting vascular wall thickening, arterial stiffness, and left ventricular hypertrophy [11, 12]. Furthermore, sympathetic nerves deeply innervate the kidneys. Excessive sympathetic outflow stimulates the juxtaglomerular apparatus, accelerating renin secretion and subsequently activating the RAAS. This inter-system crosstalk creates a classic "vicious cycle" that locks the patient into a state of chronic, treatment-resistant hypertension [13].

The strong correlation established in this study between catecholamine levels and key hemodynamic variables underscores the therapeutic necessity of targeting the sympathetic nervous system. It provides a solid pathogenetic rationale for prioritizing pharmacological interventions that blunt sympathetic outflow, such as beta-blockers or centrally acting sympatholytics (e.g., moxonidine), in the routine clinical management of these patients.

CONCLUSION

Patients diagnosed with Stage II essential hypertension exhibit profound hyperactivation of the sympathoadrenal system. This state is characterized by statistically significant elevations in plasma concentrations of both norepinephrine (55% higher than healthy controls) and epinephrine (76% higher).

A strong, positive correlation exists between circulating catecholamine levels and primary hemodynamic parameters, specifically systolic blood pressure and heart rate. This relationship confirms the leading role of sympathetic overdrive in the development, progression, and stabilization of hypertensive disease.

The documented neurohumoral shifts provide a firm pathophysiological foundation for incorporating targeted therapies aimed at restoring autonomic balance. Utilizing agents that suppress sympathoadrenal overactivity is crucial for the comprehensive and effective management of essential hypertension.

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