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THE ROLE OF THE AUTONOMIC NERVOUS SYSTEM IN THE PATHOGENESIS OF ASTHMA EXACERBATION

SUMMARY. The autonomic nervous system (ANS) plays a crucial role in regulating bronchomotor tone and airway reactivity, yet its contribution to the pathogenesis of asthma exacerbations remains insufficiently defined, particularly across eosinophilic (EA) and noneosinophilic (NEA) phenotypes.

The aim – to assess ANS activity in patients with EA and NEA during asthma exacerbation and remission using short-term heart rate variability (HRV) analysis.

Material and Methods. A total of 60 adults with asthma (30 EA, 30 NEA; aged 35–65 years) and 30 healthy controls participated in this study at Dnipro State Medical University, Ukraine. Phenotypes were classified by blood eosinophil count (EA ≥150 cells/μL; NEA <150 cells/μL). HRV parameters were derived from 5-minute ECG recordings obtained with the Polar H10 chest sensor and analyzed in Kubios HRV software. Measurements were performed during uncontrolled (UA) and controlled asthma (CA). Pulmonary function (FEV_1) and asthma control (ACQ-7) were assessed.

Results. During exacerbation, both EA and NEA groups exhibited parasympathetic predominance, reflected by high RMSSD, pNN50, and HF power, and low LF/HF ratio (EA: 0.61±0.32; NEA: 0.58±0.31). In remission, a shift toward sympathetic predominance occurred (EA: LF/HF =2.28±1.12; NEA: 2.14±1.35; $p<0.001$). No significant differences in HRV parameters were found between EA and NEA. A strong positive correlation existed between parasympathetic markers and ACQ-7 score ($\rho=0.875$), indicating that higher vagal activity was associated with poorer asthma control.

Conclusions. Asthma exacerbation in both EA and NEA phenotypes is characterized by parasympathetic dominance, which normalizes toward sympathetic activity with clinical control. Short-term HRV monitoring offers a practical, non-invasive tool for evaluating autonomic balance and disease control in asthma.

KEY WORDS: asthma; eosinophilic asthma; noneosinophilic asthma; autonomic nervous system; heart rate variability; parasympathetic activity; sympathetic balance; asthma control.

Introduction. Asthma is a chronic inflammatory disease affecting airways, with over 340 million people globally living with the condition, causing a range of respiratory symptoms like wheezing and breathlessness, with its prevalence increasing worldwide. The age-standardized incidence rate (ASIR) of asthma was predicted using the Bayesian age-cohort model, and the results suggested that 520 people per 100,000 individuals would be affected by asthma in 2050 [1]. Over the past decade, asthma has been viewed as a heterogeneous disease comprising various clinical subtypes (phenotypes) with distinct underlying mechanisms. While various approaches to asthma phenotyping exist, they all distinguish two broad pathophysiological categories, described as allergic and non-allergic asthma (based on skin prick test positivity or serum immunoglobulin E), or TH2-high and TH2-low (based on the presence/absence of TH2-inflammation or levels of blood or sputum eosinophils, fractional exhaled nitric oxide or confirmed allergy), or eosinophilic (EA) and non-eosinophilic asthma (NEA) are commonly used in research and clinical studies [2, 3]. To better understand asthma pathogenesis and optimize personalized treatment, four inflammatory asthma subtypes have been identified: EA, mixed granulocytic asthma (MGA), neutrophilic asthma (NA), and paucigranulocytic asthma (PGA), with the latter two phenotypes commonly combined into the NEA category. This approach recognizes that inflammation may not be a universal feature of asthma and that 50 % of asthma cases identified as NEA occur in the absence of allergy or eosinophilic/neutrophilic airway inflammation [4, 5].

Current understanding of the pathogenesis of both EA and NEA still contains many gaps. One such gap is the role of the autonomic nervous system (ANS). Research on this topic is limited, and the results are often unclear and contradictory. Researchers from the Institute of Medical Sciences and Research, (Tamil Nadu, India) found a strong direct correlation between PANS activity and the degree of bronchial obstruction in patients with asthma [6]. In 2023, pulmonologists from the Laboratory of Pediatric Physical Activity, Infant Center, Pontifícia Universidade Católica do Rio Grande do Sul, Porto Alegre (Brazil) published a review based on a meta-analysis of 15 studies of ANS in children and adolescents with asthma, according to which asthmatic patients were characterized by lower heart rate variability and sympathetic modulation [7]. The studies were conducted during remission; there was no separation by phenotype. Researchers from Department of

Medicine, Autonomous University of Barcelona (Barcelona, Spain) found decreased PANS activity in patients with uncontrolled asthma. PANS activity was strongly inversely correlated with the degree of airflow obstruction and anxiety levels in asthmatic patients [8]. Researchers from Massey University (Wellington, New Zealand) compared the ANS status of patients with EA and NEA during remission. They found no significant ANS imbalance in either group. The researchers attributed the increase in SNS activity found in some patients to the use of β-agonist medication. [9]. Researchers from the All India Institute of Medical Sciences (Raipur, India) found a significant predominance of parasympathetic nervous system (PANS) tone in the morning hours in asthmatic patients with nocturnal bronchospasm attacks [10]. Since nocturnal asthma attacks are more typical for NEA, these results indicate an important role of cholinergic activity in this phenotype.

It is clear that the ANS has a significant role in the pathogenesis of asthma. However, its role during different phases of the disease (exacerbation and remission) is currently unclear. It is also unclear whether its role differs in the pathogenesis of EA and NEA. These questions are particularly important given that, in accordance with current recommendations, the mainstay of inhaled therapy, along with corticosteroids, is β-agonists and muscarinic receptor agonists–medications that mediate their effects through the ANS.

The aim of the study was to investigate the ANS status in patients with EA and NEA during exacerbation and remission.

Material and Methods. This study was conducted at Dnipro State Medical University in Ukraine. We selected 60 asthmatic patients in exacerbation phase aged between 35 and 65 years, comprising 30 men and 30 women, from the allergological center in Dnipro. The control group consisted of 30 conditionally healthy individuals (healthy control subjects, HS) aged 35 to 65 years (15 men and 15 women). To facilitate asthma patient selection, we divided the participants into four randomized groups based on age and gender. All 60 patients received the same treatment regimen, which included a combination of inhaled corticosteroids (ICS) and long-acting beta-agonists (LABAs). The groups were defined as follows: the first group included patients with EA; the second group consisted of patients with NAE. A multicomponent gradient eosinophil algorithm based on expert consensus was used to distinguish EA from NEA. The algorithm uses variables readily accessible to both primary and specialist care. The main criterion for differentiation is blood eosinophil count (BEC): for EA, BEC ≥150 cells/μL (0.15×10^9/L), for NEA, BEC <150 cells/μL [11]. Lung function was assessed using a computerized spirograph (Spirolab III® MIR). Forced Expiratory Volume in 1 second (FEV1) was determined.

ANS status was assessed using Heart Rate Variability (HRV). The ANS supplies nerves to both the heart and lungs. It regulates the heart rate and the force of contraction in the heart, as well as bronchial smooth muscle constriction and mucus hypersecretion in the lungs. There is a connection between these two components. Therefore, HRV is recognized as a reliable method for diagnosing autonomic dysfunction in both the myocardium and respiratory systems. Non-invasive assessments of the ANS are conducted using HRV based on established measurement standards, physiological interpretations, and clinical uses outlined in the guidelines from the working group of the European and American Society of Cardiology and Electrophysiology [12]. In 2024, the Psychophysiological Research Societies Committee released an updated report on Heart Rate and Heart Rate Variability. This report provides guidelines for methodology, interpretation, and reporting in HRV research. It supports the use of mobile HRV recording devices and encourages the application of short-term HRV measurements [13]. Recent studies have demonstrated the validity and reliability of short-term heart rate variability (HRV) parameters in assessing the status of ANS [14, 15, 16].

HRV parameters were derived from 5-min electrocardiogram recordings by the Polar H10 sensor chest strap device (Polar Electro Oy, Kempele, Finland) in a seated position after ensuring clean ECG signals, absence of movement artifacts and comfortable breathing. After extraction of the 1-channel ECG data and RR data (exported from the Elite HRV app) import into Kubios HRV Scientific Software version 3.5.0 (Biosignal Analysis and Medical Imaging Group, Department of Physics, University of Kuopio, Finland) was conducted. For the HRV analysis, we used both the time domain and frequency domain methods: the mean heart rate (MHR), the square root of the variance of RR intervals (SDNN), the square root of the mean squared differences of successive RR intervals (RMSSD); pNN50 - the percentage of consecutive NN intervals that differ by more than 50 milliseconds, very low frequency (VLF), low frequency (LF), high frequency (HF), normalized low frequency (LF Norm), normalized high frequency (HF Norm), and the LF/HF ratio.

The study of lung function and HRV were performed during the exacerbation period (in the first 2 days of hospitalization), i.e. when asthma was uncontrolled (UA) and in dynamics after the relief of the exacerbation: after achieving control over asthma (CA), usually on the 7-10th day of treatment. Asthma

control status was based on the asthma control questionnaire (ACQ-7), with a value of ≤0.75 representing controlled asthma, 0.76–1.49 representing partially-controlled asthma, and ≥1.5 representing uncontrolled asthma [17]. The Global Initiative for Asthma recommends long-acting muscarinic antagonists (LAMAs) as additional therapy to inhaled corticosteroids (ICS) and long-acting beta-agonists (LABAs) for patients with severe asthma who are not properly managed with [18].

The study procedure was explained to the patients. We only included research participants who were willing to provide written Patient Informed Consent for the Study (Code: 22253/2024). The patients were selected based on their clinical history. We excluded patients with other diseases like hypertension, diabetes, thyroid disorders, anxiety-depressive disorders and ischemic heart disease. Smokers, alcoholics and pregnant women also were excluded from our study. All patients underwent clinical examination and investigations during the period from 09.00 AM till 12.00 PM to avoid confounding effects of circadian rhythm on HRV. On the day of the HRV study, patients did not receive inhalation therapy. Statistical evaluation was performed using the Microsoft Office Excel and IBM SPSS Statistics (version 27.0.1.0). An independent Student's t-test was used to compare the patient groups. Additionally, Spearman's correlation coefficients (ρ or r_s) were calculated to assess correlations between the variables.

Results and Discussion. Table 1 represents some characteristics of the patients with asthma. The EA and NA patient groups were randomized by gender, age, asthma severity, and prescribed therapy. The duration of asthmatic history was significantly longer in EA patients: 27.5±4.4 years versus 18.5±7.4 in the NEA group (p<0.01). This indicates an early onset of the disease, which is typical for EA [2]. In the EA group, the blood eosinophil content was almost 4 times higher than in the NEA group: 0.46±0.22 10^9/L vs. 0.12±0.08 10^9/L (p<0.001). This feature is a distinctive feature of EA. There is always a strong correlation between the eosinophil content in sputum and in peripheral blood in patients with EA [19, 20]. Upon achieving remission in the EA group, the number of eosinophils in the blood decreased by half, but was still above 0.15 10^9/L (≥150 cells/μL): 0.24±0.16 10^9/L (p<0.01), which is also typical for the course of EA. [21]. In patients with NEA, after achieving asthma control, the eosinophil level decreased slightly and insignificantly: from 0.12±0.08 10^9/L to 0.1±0.05 10^9/L (p<0.05). In the control group, the eosinophil level was 0.08±0.04 10^9/L, which is significantly lower than in both main groups during the exacerbation period (p<0.01). In patients with EA, even during remission, the eosinophil level was three times higher than in the control group (p<0.01). In patients with NEA during remission, the eosinophil level did not differ significantly from the control group (p<0.05).

The blood neutrophil level and its dynamics in the two groups of asthmatic patients did not differ significantly (p>0.05). The range of blood neutrophil levels in the NA group was significantly greater than in the EA group. This is most likely due to the presence of patients with the NA neutrophil subtype in the group [22]. However, no other significant differences were observed within the NA group. In the control group, the blood neutrophil level was normal: 2.9±1.35 10^9/L and was significantly lower than in asthmatic patients (p<0.05).

FEV1 during the exacerbation period did not differ significantly in both groups (p>0.05). After achieving asthma control, FEV1 increased by approximately 20–25 % in both groups: in patients with EA from 62.4±6.7 to 81.8±7.9% (p<0.01), and in the NEA group from 66.5±6.1 to 78.2±8.1% (p<0.01). During the remission period, there were also no significant differences in FEV1 between the groups of asthmatic patients (p>0.05). In the control group, FEV1 was 91.4±6.4%, which was 1/3 higher than in both groups of asthmatic patients during the exacerbation period (p<0.01). During the remission period, FEV1 in the main groups was on average 12.5% lower than in the control group (p<0.05) (Table 1).

Table 1. Characteristics of patients

	Eosinophilic Asthma		Noneosinophilic Asthma		Healthy control subjects
	exacerbation (UA)	remission (CA)	exacerbation (UA)	remission (CA)	
1	2		3		4
N (men/women)	30 (15/15)		30 (15/15)		30 (15/15)
Asthma duration (years)	27.5±4.4		18.5±7.4		N/A
Age (men/women)	45.6±10.2/ 48.4±12.6		52.5±11.4/ 49.3±10.6		45.6±11.2/ 48.4±13.5
Blood eosinophils, (10^9/L) Mean (range)	0.46±0.22	0.24±0.16	0.12±0.08	0.1±0.05	0.08±0.04

Continuation of Table 1

1	2		3		4
Blood Neutrophils, (10^9/L) Mean (range)	3.9±0.95	3.4±0.88	4.2±1.98	3.6±1.85	2.9±1.35
Inhaled therapy	LABA & ICS	LABA & ICS + LAMA	LABA & ICS	LABA & ICS + LAMA	
FEV1 (%)	62.4±6.7	81.8±7.9	66.5±6.1	78.2±8.1	91.4±6.4

Table 2 represents the dynamics of HRV parameters during an asthma exacerbation and after achieving remission, i.e., after asthma control was achieved. The analysis shows that in both groups, the ANS status during the exacerbation was characterized by a predominance of PANS activity, whereas during remission, both groups demonstrated a predominance of the sympathetic ANS (SANS).

Table 2. HRV in asthmatic patients during exacerbation and remission

	Eosinophilic Asthma		Noneosinophilic Asthma		Control group (healthy persons)
	exacerbation (Uncontrolled asthma)	remission (Controlled asthma)	exacerbation (Uncontrolled asthma)	remission (Controlled asthma)	
MHR (bpm)	62.4±6.5	89.1±8.4	61.3±7.6	87.6±9.7	72.4±7.1
SDNN, (ms)	23.5±8.7	46.5±15.7	21.5±7.6	44.2±11.8	55.5±16.2
pNN50 (%)	8.32±2.16	0.74±0.25	9.54±2.12	0.68±0.32	3.52±1.24
RMSSD, (ms)	48.5±15.8	22.7±5.5	46.4±16.7	21.5±6.8	35.6 ±12.6
LF (ms^2)	323.6±178.2	852.5±287.7	315.6±158.5	839.2±186.3	522.6±204.7
HF (ms^2)	934.2±321.4	413.6±134.1	898.6±379.4	435.6±153.8	648.6±225.8
LF Norm (n.u., normalized units)	36.3±15.8	58.7±22.6	34.5±17.4	61.4±20.6	44.7 ± 12.5
HF Norm (n.u.)	55.4±16.2	31.6 ± 12.7	57.6±18.5	28.5±14.2	41.3±10.2
LF/HF Ratio	0.61±0.32	2.28±1.12	0.58±0.31	2.14±1.35	1.58±0.78
ACQ-7	4.58±0.65	0.55±0.15	4.27±0.75	0.6±0.15	N/A

During the exacerbation period, MHR showed a tendency toward bradycardia: 62.4±6.5 beats per minute (bpm) in the EA group and 61.3±7.6 bpm in the NEA group (p>0.05). After asthma control was achieved, MHR in patients of both groups showed a tendency toward tachycardia: 89.1±8.4 in EA patients and 87.6±9.7 bpm in the NEA group (p>0.05). In the control group, MHR was 72.4±7.1 bpm, which was significantly higher than in both main groups during the exacerbation period (p<0.05) and lower than during the remission period (p<0.05) (Table 1).

Decreased SDNN during the exacerbation period in both groups of asthmatic patients indicates a decrease in the adaptive capacity of the ANS: 23.5±8.7 ms in patients with EA and 21.5±7.6 in patients with NEA (p>0.05). During remission, SDNN increased to normal in both groups: 46.5±15.7 in patients with EA and 44.2±11.8 in the NEA group (p>0.05) [13, 23]. SDNN in the control group was 2.5 times higher than in both groups of asthmatic patients in the exacerbation phase: 55.5±16.2 (p<0.01), whereas in the remission phase this difference was insignificant (p>0.05).

The pNN50 reflects the activity of the PANS, indicating the degree of vagal tone. A higher pNN50 value generally suggests greater parasympathetic activity. pNN50 is closely correlated the RMSSD and HF power [13, 23]. In patients with EA, pNN50 indicated a marked predominance of PANS activity during the exacerbation period: 8.32±2.16. Upon achieving asthma control, it indicated a moderate increase in SANS activity: 0.74±0.25 (p<0.001). In the NEA group, pNN50 demonstrated similar dynamics: 9.54±2.12 during the exacerbation period and 0.68±0.32 in remission (p<0.001). There were no significant differences between the EA and NEA groups (p>0.05). In the control group, pNN50 was 3.52±1.24, which is characteristic of a balanced ANS state [23]. The pNN50 in the control group was 2.5 times lower than in both main groups during the exacerbation period (p<0.001) and 5 times higher than during the remission period (p<0.001).

The RMSSD reflects the beat-to-beat variance in HR and is the primary time-domain measure used to estimate the vagally mediated changes reflected in HRV. The higher the RMSSD, the higher the PANS activity. The RMSSD in the groups of asthmatic patients during the exacerbation period indicated the predominance of cholinergic activity: in EA patients

48.5±15.8 ms, and in NEA patients 46.4±16.7 ms (p>0.05). Upon achieving asthma control, RMSSD significantly and reliably decreased (p<0.001) to 22.7±5.5 ms and to 21.5±6.8 ms (p>0.05), respectively, indicating a moderate predominance of adrenergic activity. In the control group, RMSSD was 35.6±12.6 ms, indicating a balanced PANS status (p<0.05) [13, 23].

LF indicates activity of the sympathetic modulators. HF component HRV indicates vagal efferent activity, the higher the HF value, the greater the activity of the PANS. The benefit of LF Norm and HF Norm is that they enable comparisons among individuals by reducing variations in overall HRV. LF/HF ratio characterizes the sympathetic-vagal balance [23]. LF power (absolute and normalized) demonstrated a change in SANS activity in EA and NEA patients from decreased during the exacerbation period (respectively 36.3±15.8 n.u. and 34.5±17.4 8 n.u., p>0.05) to increased during the remission period, p<0.001 (respectively 58.7±22.6 8 n.u. and 61.4±20.6 n.u., p 0.05). HF power (absolute and normalized) in EA and NEA patients demonstrated a reliable change in ANS cholinergic activity from high during the exacerbation period (55.4±16.2 n.u. and 57.6±18.5 n.u., respectively, p>0.05) to low during remission, p<0.001 (31.6± 12.7 n.u. and 28.5±14.2 n.u., respectively, p>0.05). In the control group, LF power and HF power significantly differed from the indicators of both groups of asthmatic patients (p<0.05) and corresponded to a balanced state of ANS: LF Norm 44.7±12.5 n.u., HF Norm 41.3±10.2 n.u.

LF/HF Ratio in the main groups during the exacerbation period indicated a marked increase in vagal tone: 0.61±0.32 in EA patients and 0.58±0.31 in NEA patients (p<0.05). During the remission period, LF/HF Ratio significantly and reliably (p<0.01) increased to 2.28±1.12 in EA patients and to 2.14±1.35 in NEA patients (p<0.05), which corresponded to the predominance of SANS activity [13, 23]. In the control group, LF/HF Ratio was 1.58±0.78 and corresponded to the normotonic state of ANS. The dynamics of LF/HF Ratio in EA and NEA patients is presented in Fig. 1.

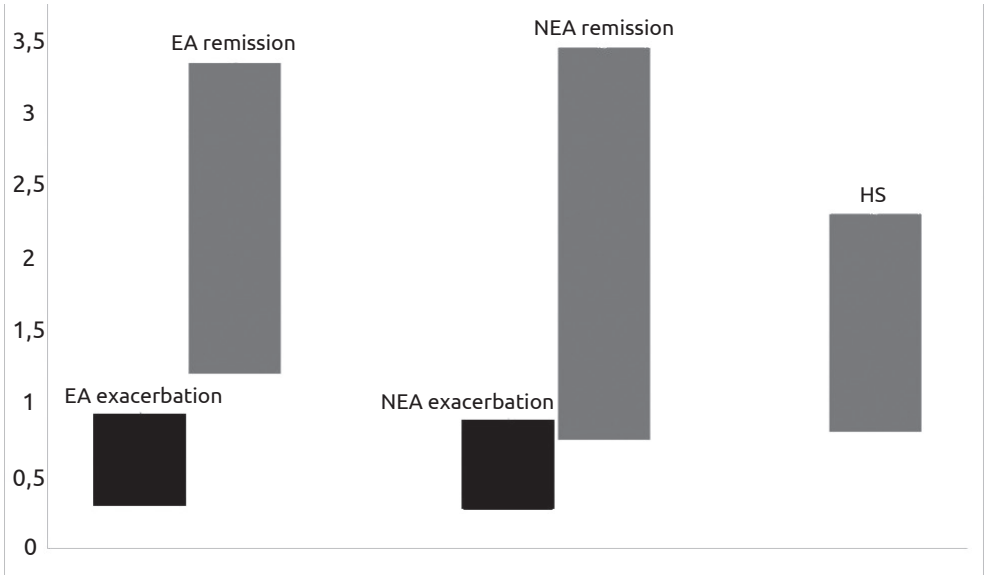

Fig. 1. LF/HF Ratio in asthmatic patients and in control group.

Thus, the ANS of patients with EA and NEA demonstrated a marked predominance of cholinergic activity during an exacerbation, which changed to a moderate predominance of adrenergic activity when asthma control was achieved. Spearman's coefficient indicated a strong direct correlation between HRV parameters related to parasympathetic tone and the Overall Score on the ACQ7: the higher the pNN50 (ρ=0.812), RMSSD (ρ=0.785), HF (ρ=0.615), and HF Norm (ρ=0.835), the higher the Overall Score on the ACQ7. In other words, the higher the PANS activity, the worse the asthma control. Fig. 2 shows the correlation between the LF/HF Ratio and the Overall Score on the ACQ7 (ρ=0.875).

When analyzing the HRV assessment results, we attempted to minimize the influence of inhalation therapy: patients refrained from using ICS, LABA or LAMA for several hours before the study. However, patients with uncontrolled asthma were allowed to use salbutamol. The existing data on the effects of inhaled LABA and LAMA on HRV are contradictory. For instance, some researchers found that LABA and LAMA do not significantly affect HRV in asthma patients [24]. In contrast, others reported that the administration of salbutamol within two hours prior to HRV testing or the use of LABA within two weeks prior resulted in decreased PANS activity and increased SNS activity in asthma patients [25]. The

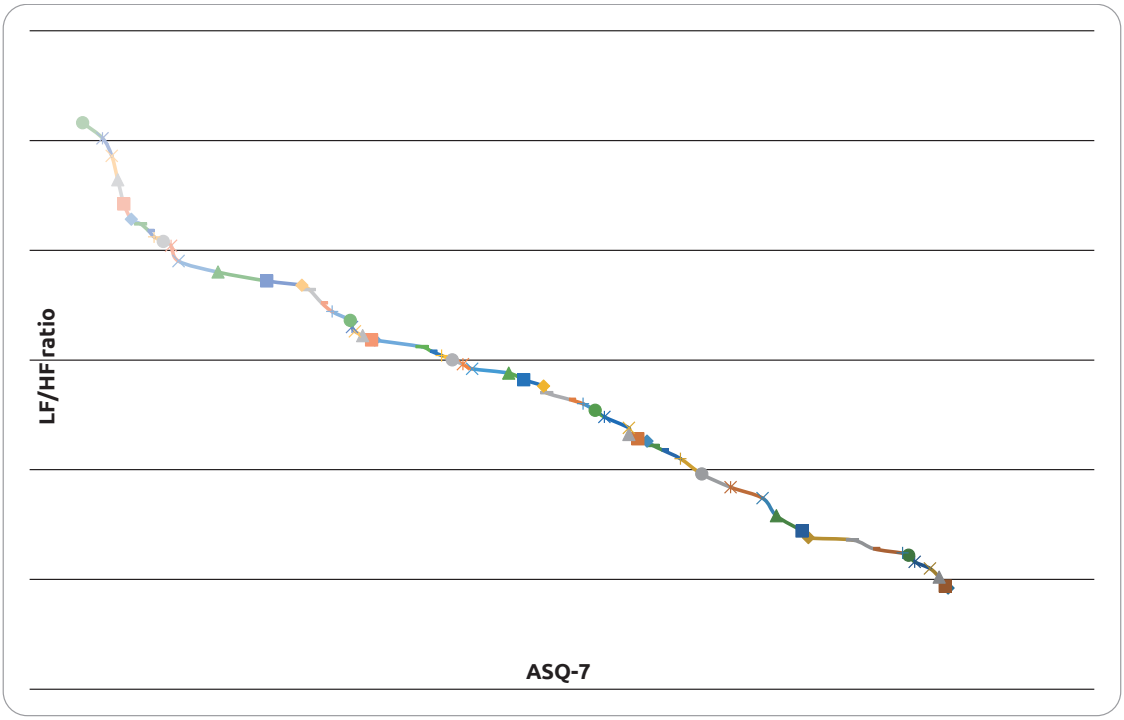

Fig. 2. Correlation between LF/HF Ratio and the Overall Score on the ACQ7 (ρ = 0.875)

researchers also provided evidence that long-acting β2-agonists caused a very long-lasting increase in SNS activity [26].

In the present study, all asthmatic patients received a combination of ICS and LABA as inhalation therapy during an exacerbation, which might be expected to increase SANS activity. However, patients with EA and NEA demonstrated a significant predominance of PANS activity during this period. This eliminates any doubt about the predominance of parasympathetic activity in asthmatic patients during the exacerbation phase. If LABA did have an effect, it was offset by the robust PANS activity.

In accordance with current guidelines for stepped inhalation therapy, all asthmatic patients were given LAMAs in addition to their inhalation therapy. LAMAs reduce vagus activity by blocking acetylcholine's action at muscarinic receptors, which are activated by the parasympathetic nervous system, including the vagus nerve. This action interrupts vagal signals that cause bronchoconstriction and smooth muscle contraction, leading to airway relaxation and bronchodilation [18].

Patients with EA and NEA did enter remission after adding LAMA to their inhalation therapy and demonstrated a significant reduction in PANS activity, resulting in a moderate predominance of SANS activity. The causal relationship between ANS status and asthma control remains unclear: does relieving asthma exacerbations lead to SANS predominance, or does suppressing PANS activity lead to asthma control?

Conclusion. 1. No differences were found between EA and NEA in ANS status.

2. During an exacerbation (lack of asthma control), the ANS of patients with EA and NEA is characterized by a predominance of PANS activity, while during remission (achieving asthma control), the ANS demonstrates a predominance of SANS activity.

3. The strong correlation between the degree of asthma control and ANS status allows us to recommend assessing ANS status in a comprehensive evaluation of patients with asthma.

4. The short-term HRV is a convenient and accurate method for assessing ANS. The method does not require additional expensive equipment and is quick to use, making it recommended for use by primary care physicians and family physicians.

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Author`s contribution:

Iryna Vysochyna – conceptualization, methodology, proofreading of the article.

Ivan Berezutskyi – research, resources, data curation, software.

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Declaration on the use of artificial intelligence. No AI was used in the preparation of the manuscript.

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РОЛЬ АВТОНОМНОЇ НЕРВОВОЇ СИСТЕМИ В ПАТОГЕНЕЗІ ЗАГОСТРЕННЯ БРОНХІАЛЬНОЇ АСТМИ

РЕЗЮМЕ. Автономна нервова система (АНС) відіграє ключову роль у регуляції бронхомоторного тонусу та реактивності дихальних шляхів, однак її внесок у патогенез загострень бронхіальної астми досі залишається недостатньо з'ясованим, особливо з огляду на різні фенотипи – еозинофільний (ЕА) та не-еозинофільний (NEA).

Мета – оцінити активність АНС у пацієнтів з ЕА та NEA під час загострення та ремісії астми за допомогою короткочасного аналізу варіабельності серцевого ритму (ВСР).

Матеріал і методи. У дослідженні, проведеному на базі Дніпровського державного медичного університету (Україна), взяли участь 60 пацієнтів із астмою (30 ЕА, 30 NEA; вік 35–65 років) та 30 практично здорових осіб. Фенотипи визначали за рівнем еозинофілів у крові (ЕА ≥150 кл/мкл; NEA <150 кл/мкл). Показники ВСР отримували з 5-хвилинних ЕКГ-записів, зареєстрованих сенсором Polar H10, і аналізували у програмі Kubios HRV. Обстеження проводили під час неконтрольованої астми (UA) та після досягнення контролю (CA). Додатково оцінювали функцію зовнішнього дихання ($ОФВ_1$) і рівень контролю астми (ACQ-7).

Результати. У період загострення обидві групи продемонстрували переважання парасимпатичної активності, що проявлялося високими значеннями RMSSD, pNN50 та HF і низьким співвідношенням LF/HF (ЕА: 0,61±0,32; NEA: 0,58±0,31). У ремісії спостерігалося зміщення у бік симпатичної домінанти (ЕА: LF/HF=2,28±1,12; NEA: 2,14±1,35; p<0,001). Значущих відмінностей між ЕА та NEA не виявлено. Встановлено сильну пряму кореляцію між показниками парасимпатичної активності та сумарним балом ACQ-7 (ρ=0,875), що свідчить: чим вища парасимпатична активність, тим гірший контроль астми.

Висновки. Загострення бронхіальної астми у пацієнтів обох фенотипів характеризується переважанням парасимпатичної активності, яка нормалізується до симпатичної при досягненні клінічного контролю. Короткочасний аналіз ВСР є зручним неінвазивним методом оцінки автономного балансу та стану контролю бронхіальної астми.

КЛЮЧОВІ СЛОВА: бронхіальна астма; еозинофільна астма; не-еозинофільна астма; автономна нервова система; варіабельність серцевого ритму; парасимпатична активність; симпатичний тонус; контроль астми.

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