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Роль нейропептида галанина в патогенезе формирования позных нарушений при болезни Паркинсона

Н. В. Селянина[1]✉, Ю. В. Каракулова[1], О. В. Хегай[2]

[1] Пермский государственный медицинский университет имени академика Е. А. Вагнера, Пермь, Российская Федерация;
[2] Институт мозга человека имени Н. П. Бехтеревой Российской академии наук, Санкт-Петербург, Российская Федерация

АННОТАЦИЯ

Введение. Позные нарушения при болезни Паркинсона (БП) являются симптомами, патогенез которых остается малоизученным, а ранняя объективная диагностика затруднена. В литературных источниках отмечено влияние нейромодулятора галанина на многие структуры центральной и периферической нервной системы, что указывает на возможность его участия в формировании моторных нарушений при БП.

Цель. Изучить содержание нейромодулятора галанина в сыворотке крови у пациентов с БП и определить его влияние на клинические проявления позных нарушений.

Материалы и методы. Обследовано 73 пациента с диагнозом БП, группа контроля — 22 здоровых волонтёра. Оценивались жалобы, анамнез, неврологический статус. В основной группе выявлены «классические» жалобы и клинические проявления. Использовались шкала Хен и Яр, унифицированная шкала оценки при БП. Содержание галанина в сыворотке определялось методом иммуноферментного анализа.

Результаты. Стадия заболевания составила в среднем 2 [2; 3], тяжесть двигательного дефицита по унифицированной шкале оценки БП — 46 [36; 56] баллов. У 8 пациентов зафиксированы тяжёлые расстройства позы; у 21 — умеренные, лёгкие — у 28 человек, очень лёгкие — у 8 пациентов. В основной группе уровень галанина составил 6,0 [4,2;10,3] пг/мл, что значительно ниже, чем в группе контроля (16,9[9,8; 18,1] пг/мл; p = 0,001). Корреляционный анализ выявил взаимосвязь уровня галанина и выраженности позных расстройств (R = -0,73; p = 0,001). При содержании галанина выше 26 пг/мл нарушения позы у пациентов отсутствовали, 8–26 пг/мл соответствовали минимальным и лёгким нарушениям, 5–8 пг/мл — умеренным, менее 5 пг/мл — тяжёлым.

Заключение. Снижение уровня сывороточного галанина сопряжено со степенью выраженности позных расстройствпри болезни Паркинсона.

Ключевые слова: *болезнь Паркинсона; двигательные нарушения; позные расстройства; камптокормия; галанин*

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Role of Galanin Neuropeptide in Pathogenesis of Postural Disorders in Parkinson's Disease

Nataliya V. Selyanina[1]✉, Yuliya V. Karakulova[1], Ol'ga V. Khegay[2]

[1] E. A. Vagner Perm State Medical University, Perm, Russian Federation;
[2] N. P. Bekhtereva Institute of the Human Brain of the Russian Academy of Sciences, Saint-Petersburg, Russian Federation

ABSTRACT

INTRODUCTION: Postural disorders in Parkinson's disease (PD) are symptoms with poorly understood pathogenesis, and the early objective diagnosis is difficult. In the literature, the influence of galanin neuromodulator on many structures of peripheral and central nervous system has been described, which indicates the probable involvement of the nervous system in the formation of motor disorders in PD.

AIM: To study the content of galanin neuromodulator in blood serum of patients with PD and to determine its effect on clinical manifestations of postural disorders.

MATERIALS AND METHODS: Seventy-three patients with PD diagnosis were examined, the control group included 22 healthy volunteers. The complaints, history and neurological status were evaluated. In the main group, 'classic' complaints and clinical manifestations were identified. The Hoehn and Yahr unified rating scale for PD was used. The content of galanin in blood serum was determined using the enzyme-linked immunoassay.

RESULTS: The stage of the disease on average was 2 [2; 3], severity of motor deficit on the unified PD assessment scale was 46 [36; 56] points. In 8 patients, severe postural disorders were identified, moderate disorders — in 21, mild disorders — in 28 patients, very mild — in 8 patients. The level of galanin in the main group was 6.0 [4.2; 10.3] pg/ml, which is considerably lower than in the control group (16.9 [9.8; 18.1] pg/ml; p = 0.001). The correlation analysis revealed the relationship between the level of galanin and severity of postural disorders (R = -0.73; p = 0.001). With the content of galanin above 26 pg/ml, no postural disorders were present in the patients, the content 8 pg/ml — 26 pg/ml was associated with the minimal and mild disorders, 5 pg/ml – 8 pg/ml — with moderate disorders, and the content below 5 pg/ml — with severe disorders.

CONCLUSION: Reduction of serum galanin level is associated with the degree of severity of postural disorders in Parkinson's disease.

Keywords: *Parkinson's disease; motor disorders; postural disorders; camptocormia; galanin*

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ECO•VECTOR

LIST OF ABREVIATIONS

PD — Parkinson's disease
UPDRS — Unified Parkinson's disease rating scale

INTRODUCTION

Postural disorders are a characteristic phenomenon in Parkinson's disease (PD), they may occur at any stage of the disease but most often appear as the disease progresses [1, 2]. The most common postural deformity in PD is a change of the normal posture of the patient with flexion of the head and trunk forward, sideward or as a scoliosis-like trunk deformation [3]. The severity of these disorders can vary from a mild degree where a change in the posture can be attributed to the age, to severe in the form of camptocormia ('flexed back' syndrome, Figure 1), camptocephalia ('bent head' syndrome), lateral flexion of the trunk (Pisa syndrome) [4]. Severe manifestations in the form of camptocormia are less common, however, their prevalence among the patients with PD ranges from 3% to 17% [1, 4].

Fig. 1. A postural disorder in Parkinson's disease in the form of camptocormia — 'flexed back' syndrome (observation of authors).

The pathogenetic mechanisms of postural disorders remain poorly understood [5]. Some authors [6] consider camptocormia to be an extreme manifestation of rigidity of the axial muscles, leading to tonic flexion of the trunk. In another version, severe postural deformities are interpreted as dystonic phenomena [7]. Besides, a mechanism is also considered based on the specific myopathy of the muscles involved in the antigravity processes [8].

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An important task is timely detection of postural deformities, since these manifestations of the disease significantly reduce the quality of patients' life. However, the early objective diagnosis of these disorders with the existing methods is difficult. In most cases, patients do not present with active complaints of changes in posture, this manifestation is ignored, or is regarded as a degenerative-dystrophic process of the musculoskeletal system [8]. In this context, an important task is the search for additional methods of stating postural disorders and verifying their severity.

According to Z Wu, et al. (2014), PD is associated with a powerful production of galanin in the tuberomammillary nucleus of the hypothalamus, which has a modulating effect on the motor neurons of the spinal cord and cerebellum [9]. Galanin is a neuromodulator with the action mediated by three types of galanin receptors found in many structures of the central and peripheral nervous system, including the basal ganglia, neocortex, Meynert nucleus, olfactory bulb, *colliculus inferior*, and blue spot [10]. These factors formed the basis of our assumption about the relationship of galanin with the formation of a pathological posture in PD.

The **aim** was to study serum galanin content in patients with Parkinson's disease and determine its effect on the clinical manifestations of postural disorders.

MATERIALS AND METHODS

The work was performed in accordance with the ethical principles of the World Medical Organization's Declaration of Helsinki 'Ethical Principles for Medical Research Including Human Subjects' and was approved by the Ethics Committee of E. A. Vagner Perm State Medical University (Protocol No. 11 of December 27, 2017). All the included subjects signed informed consent to participation in the study.

On the basis of Perm Regional Center for the Diagnosis and Treatment of Extrapyramidal Disorders, 95 patients were examined, 73 of whom (39 women and 34 men) were diagnosed with PD without decompensated concomitant pathology and were included in the main group. The age of patients of the main group ranged from 43 to 79 years, and was on average 68.1 [63; 74] years.

The patients with PD were distributed by stages of the disease as follows: stage 1 — 18 people, stage 2 — 20, stage 3 — 26, stage 4 — 9; 34 patients had a tremor-rigid form of the disease, 6 — akinetic-rigid form, 12 — rigid-tremor, 17 — akinetic-rigid-tremor form. Fourteen patients in the main group were newly diagnosed with PD and have not undergone antiparkinsonian therapy before the study, 59 people of the main group received therapy in accordance with domestic PD treatment protocols [4].

The control group, comparable in gender ($p = 0.42$) and age ($p = 0.43$), included 22 practically healthy individuals who turned to a neurologist as part of periodical medical examination of the population.

The clinical examination included study of complaints, history, and assessment of neurological status. The stage of the disease was determined on the classic Hoehn and Yahr scale. The neurological deficit was quantified on the Unified Parkinson's disease rating scale (UPDRS) [11]. The motor deficit was assessed on both sides; in patients with 2–4 stage PD according to Hoehn and Yahr, having bilateral symptoms, the symptoms of the dominant side were taken into account in statistical calculations.

The quantitative content of galanin neuropeptide in blood serum was determined by competitive enzyme immunoassay. The CEB084Hu ELISA Kit test systems for galanin (Cloud-Clone Corp, USA) were used. Galanin content in blood serum of patients of the main group was compared to the parameters of the control group.

The data obtained were processed in the Statistica 10.0 program (Stat Soft Inc., USA). The median, standard deviation, and interquartile range (25%, 75%) were calculated using descriptive statistics methods. The Mann–Whitney U-test was used to compare two independent samples of nonparametric data. A threshold of $p < 0.05$ was used to assess the probability of validity of the null hypothesis for the two groups. To identify the relationship of various factors, Spearman's correlation coefficient was used; the critical level of significance when testing statistical hypotheses was considered equal to 0.05. To model the hypotheses and assess the contribution of individual independent factors to the development of an attribute and predict its development, the regression analysis method was used.

RESULTS

Patients of the main group presented with 'classic' complaints: resting tremor (n = 67), rigidity (n = 70), slowness of movement (n = 73), impaired fine motor skills (n = 61), gait changes (n = 66), less often a change in erectness (n = 49), falls (n = 4), decrease in the sonority of the voice (n = 9). In the light of the study of postural disorders in PD, it is important to emphasize that some patients (n = 39) complaining of impairment of erectness noted that it was with this symptom that the development of the disease began (followed by the addition of other motor disorders), but this did not become a reason to consult a specialist.

Examination revealed characteristic motor disorders in most patients with PD: hypokinesia (n = 71), resting tremor (n = 68), rigidity (n = 71), postural instability

(n = 33). In many patients of the main group (n = 65), a forward flexion of the head and/or trunk was detected, in some cases accompanied by fixation of the limbs in a slightly flexed position with the formation of a 'supplicant posture'. Severe postural disorders (camptocormia, Pisa symptom) were detected in 8 cases. In general, the UPDRS score of the average severity of these disorders was 2 [2; 3] points, which corresponds to a mild degree. The severity of the disease on the Hoehn and Yahr scale was also estimated 2 [2; 3] points. The average score in the second part of UPDRS (motor aspects of everyday life) turned out to be 33 [24; 49], and the quantitative assessment of objective motor deficit in part 3 of UPDRS was 46 [36; 56] points. The severity of motor complications on UPDRS ranged from 1 to 10 points (n = 36), averaging 4 [2; 6] points.

Distribution of postural disorders by severity was as follows: severe degree (n = 8) was manifested by the evident change in the posture in the form of forward flexion, scoliosis or lateral flexion; moderate degree (n = 21) was manifested by a forward flexion, scoliosis or bend which could not be voluntarily corrected by a patient; mild degree (n = 28) was a tilt of the head forward or to the side, that could not be overcome by an effort of will; very mild degree (n = 8) by not very erect posture, which, however, can be considered normal for an elderly person. To note, no correlations were found between postural disorders and PD staging. In comparison of the degree of severity of postural disorders with other motor manifestations, a mild direct correlation dependence was identified with the following symptoms:

- arm rigidity (R = 0.23; p = 0.04);
- gait disorder (R = 0.27; p = 0.02);
- postural instability (R = 0.27; p = 0.01);
- resting tremor amplitude (R = 0.49; p = 0.001),

which in general indicates the association of postural disorder with impairment of tone, postural dysfunction and impairment of walking.

The content of galanin in blood serum of patients with PD ranged from 1.2 pg/ml to 56.8 pg/ml (on average 6.0 [4.2; 10.3] pg/ml). In the control group, the level of galanin appeared to be considerably higher than in the main cohort of patients, averaging 16.9 [9.8; 18.1] pg/ml, p = 0.001.

Correlation analysis showed an inverse dependence of the quantitative galanin content on the severity of postural disorders (R = -0.73; p = 0.001). It is worth noting that the parameters of the studied neuropeptide were highest in patients without clinical manifestations of postural disorders (26.9 [16.4; 33.8] pg/ml). At the same time, in patients with minimal manifestations of postural deformities, the quantitative content of galanin was different from the norm (8.2 [4.6; 18.5] pg/ml, p = 0.01).

Patients of the main group with minimal and mild (8.9 [4.8; 10.3] pg/ml) postural disorders did not differ in the content of humoral galanin, p = 0.06. However, in the group of patients with moderate severity of these symptoms, the level of the studied neuropeptide was lower (4.4 [3.9; 5.5] pg/ml; p = 0.004) compared with mild postural disorders. Patients with severe postural deformities had even lower galanin levels (2.0 [1.2; 3.3] pg/ml) compared to patients with moderate impairments (p = 0.001). The dependence of the quantitative content of galanin and severity of postural disorders is presented in Figure 2 and Table 1.

Table 1. Content of Galanin in Blood Serum of Patients with Different Degree of Postural Disorders in Parkinson Disease

Degree of Severity of Postural Disorders	Serum Galanin Level, pg/ml
No postural disorders	26.9 [16.4; 33.8]
Very mild	8.2 [4.6; 18.5]
Mild	8.9 [4.8; 10.3]
Moderate	4.4 [3.9; 5.5]
Severe	2.0 [1.2; 3.3]

The regression analysis revealed that the serum galanin content > 26 pg/ml clinically corresponded to the absence of a pathological posture, while the range of 8 pg/ml–26 pg/ml was determined in patients with minimal and mild postural disorders, 5 pg/ml–8 pg/ml with moderate disorders, and the level < 5 pg/ml was recorded in PD with severe postural deformations.

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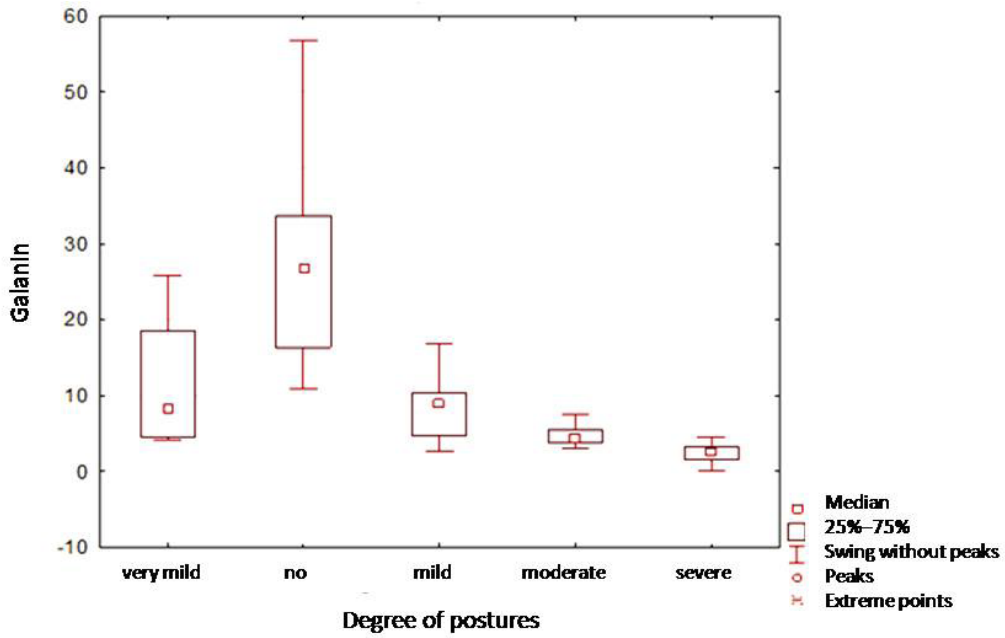

Fig. 2. Graphic presentation of content of galanin in blood serum (Y axis; pg/ml) of patients with different degree of postural disorders in Parkinson disease (X axis).

DISCUSSION

The dependence of the severity of postural disorders on the quantitative content of serum galanin, in our opinion, is associated with the modulating effect of the neuropeptide on the tonic activity of spinal motor neurons. This assumption is based on the data from Z Wu, et al. (2014) on the galanin-ergic modulating effect of the tuberomammillary nucleus of the hypothalamus on the cerebellum and motor neurons of the spinal cord. Thus, in a decrease in galanin concentration, the glutamate-ergic system is activated, and increase in the tonic activity from the tuberomammillary nucleus of spinal neurons along the descending tract leads to the development of axial dystonia and the formation of pathological postures [9]. The hypothesis of central, but non-dopaminergic, neurotransmitter mechanisms of camptocormia is confirmed by the low effectiveness of levodopa preparations for its correction [12]. The idea of the central mechanism of development of camptocormia of the axial dystonia type is supported by a decrease in the severity of postural disorders when using 'sensory tricks' in some cases, including sensory biofeedback to direct the voluntary attention to correction of posture [13]. To that end, a decrease in the tone of the paravertebral muscles is due to their overload during compensatory erecting the trunk. This hypothesis is confirmed by the sufficiently effective use of special orthoses manufactured according to the principle of thoracic-pelvic anterior fixation to alleviate camptocormia [14].

Summarizing the results of the literature analysis and of our study, it should be concluded that the presented mechanisms of development of postural disorders in PD complement each other and are factors of the same chain. Participation of galanin-ergic system is fairly justified and has been proven by a high degree of correlation dependence between the severity of postural disorders in patients with PD and quantitative content of humoral galanin. The conducted study suggested a possibility of using laboratory determination of serum galanin as a marker of early objective diagnosis of pathological postures in patients with PD and determination of their degree of severity.

CONCLUSION

Thus, the data obtained confirm the suggestion about the involvement of galanin-ergic system in the pathogenesis of pathological postures. Reduction of serum galanin in patients with Parkinson's disease is associated with enhancement of postural disorders.

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ОБ АВТОРАХ

***Селянина Наталия Васильевна**, д.м.н., доцент;
ORCID: https://orcid.org/0000-0002-2317-7808;
eLibrary SPIN: 9379-1027; e-mail: nselyanina@mail.ru

Каракулова Юлия Владимировна, д.м.н., профессор;
ORCID: https://orcid.org/0000-0002-7536-2060;
eLibrary SPIN: 5066-6556; e-mail: julia.karakulova@mail.ru

Хегай Ольга Викторовна, к.м.н.;
ORCID: https://orcid.org/0000-0001-9510-239X;
eLibrary SPIN: 5729-3467; e-mail: gamletslemon@yandex.ru

AUTHOR'S INFO

***Nataliya V. Selyanina**, MD, Dr. Sci. (Med.), Associate Professor;
ORCID: https://orcid.org/0000-0002-2317-7808;
eLibrary SPIN: 9379-1027; e-mail: nselyanina@mail.ru

Yuliya V. Karakulova, MD, Dr. Sci. (Med.), Professor;
ORCID: https://orcid.org/0000-0002-7536-2060;
eLibrary SPIN: 5066-6556; e-mail: julia.karakulova@mail.ru

Ol'ga V. Khegay, MD, Cand. Sci. (Med.);
ORCID: https://orcid.org/0000-0001-9510-239X;
eLibrary SPIN: 5729-3467; e-mail: gamletslemon@yandex.ru

* Автор, ответственный за переписку / Corresponding author