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PARKINSON KASALLIGIDA KLINIK SIMPTOMLARNI SHAKLLANISHIDA IMMUNOLOGIK OMILLARNING ROLI

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Annotatsiya. *Parkinson kasalligi neyrodegenerativ kasallik bo‘lib, uning klinik simptomlarini shakllanishida turli xil omillar, jumladan immunologik mexanizmlar muhim rol o‘ynaydi. Ushbu maqolada Parkinson kasalligida immunologik omillarning neyronlarga ta’siri, autoimmun javoblar va yallig‘lanish jarayonlari ko‘rib chiqiladi. Tadqiqot immunologik omillarni tushunish orqali davolash strategiyalarini takomillashtirishga yordam beradi.*

Kalit so'zlar*: Parkinson kasalligi, immunologik omillar, neyrodegeneratsiya, autoimmun javob, yallig‘lanish jarayonlari, klinik simptomlar.*

РОЛЬ ИММУНОЛОГИЧЕСКИХ ФАКТОРОВ В РАЗВИТИИ КЛИНИЧЕСКИХ СИМПТОМОВ ПРИ БОЛЕЗНИ ПАРКИНСОНА

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Аннотация. *Болезнь Паркинсона — это нейродегенеративное заболевание, в развитии клинических симптомов которого значительную роль играют различные факторы, включая иммунологические механизмы. В данной статье рассматривается влияние иммунологических факторов на нейроны, аутоиммунные реакции и воспалительные процессы при болезни Паркинсона. Исследование способствует улучшению стратегий лечения за счёт понимания иммунологических процессов.*

Ключевые слова: *Болезнь Паркинсона, иммунологические факторы, нейродегенерация, аутоиммунные реакции, воспалительные процессы, клинические симптомы.*

THE ROLE OF IMMUNOLOGICAL FACTORS IN THE DEVELOPMENT OF CLINICAL SYMPTOMS IN PARKINSON'S DISEASE

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Abstract. *Parkinson's disease is a neurodegenerative disorder in which various factors, including immunological mechanisms, play a significant role in the development of clinical symptoms. This article explores the impact of immunological factors on neurons, autoimmune responses, and inflammatory processes in Parkinson's disease. The study aims to enhance treatment strategies by improving the understanding of immunological aspects.*

Keywords: *Parkinson's disease, immunological factors, neurodegeneration, autoimmune responses, inflammatory processes, clinical symptoms.*

Parkinson's disease (PD) is chronic and little by little the climax receiver neurodegenerative disease mostly black substance (substantia nigra) dopaminergic of neurons degeneration with depend Clinical in terms of PK hypotension and of muscles rigidity, tremor and condition Disruptions are also different different discontinuous symptoms with manifestation will be [1].

Last ten PK pathogenesis in the year in learning disease development immunological mechanisms separately attention is focused [2]. Especially

neurodegeneration in the process of pro- and to inflammation against of cytokines participation big important have the fact that is emphasized [3, 4]. in PK cytokine situation study for 10 years more than time during take going, however long time continue reached studies and many clinical-experimental to work despite, in PK blood and back the brain different in liquid (OMS). of cytokines concentration about data opposite and scattered to the feature have

For example, in 1996 with PK it hurt of patients in the blood level of interleukins (IL-1 b, IL-2, IL-6). learned and only of the level of IL-2 increased determined. Other to information on the contrary, with mitogen stimulation from being done then of PK patients blood IL-2 production in monocytes release decreased, IL-1 b, IL-6 and tumor necrosis factor alpha (TNF α) level increase observed [6]. From this besides, there are many studies authors of the disease duration, stage and clinical of appearances weight account did not get Therefore, cytokine degrees and disease manifestation to be between dependence to determine current is considered This in the field only some studies available [9, 8].

For example, D. Lindqvist and associates [7] with PD it hurt of patients IL-6 and TNFα in the blood level increase worry and depression diagnostic to scales according to their weight level with positive dependence determined. Nevertheless, blood, especially in OMS cytokine profile PD stage of indicators of the disease start age, development speed, available of disorders lateralization, movement and motionless of symptoms weight with dependence is still an open question remains

Materials and methods. Research on Parkinson's disease with 90 people were injured patient (49 people a woman and 41 people male) attraction done Theirs disease stage Henn and Yarning in detail to the scale 1.0 to 4.0 respectively was Diagnosis " Idiopathic Parkinson's disease diagnosis to the " do " criteria according to done increased Patients aged 43 to 82 years average age 65 (57-73) years was In research participation of those who have reached main portion from 1.5 to 2.5 was stage patients was (65 people, 72%).

Illness development pace

- Rapid development pace: 17 people patient (19%).
- Average pace: 52 people patient (58%).
- Slow down development: 21 people patient (23%).

Illness duration from 2 to 16 years, mostly 5-9 years during disease with who lived superiority did (39.1%). Illness the beginning age from 33 to 76 years, median and 57.7 (51-67) years old was It's early from the age of 45 with initial PK before disease 7 people started patient (8.1%) noted done

Control group Control group to the composition aged 46 to 75 years 25 people who have person (14 people a woman and 11 people male) is included. They are from illness free is, age and sex in terms of PK patients with coordinated.

Research methods

- Clinical and neurological evaluation.
- The following scales using assessment:

○ Parkinson's disease unified rating scale (UPDRS),

○ Schwab and of England diary activity scale,

○ NMS (clinical symptoms),

○ No depression scale and another tests.

Biological of samples analysis. Patients blood (90 PD patients and 25 people control group) and 16 patients back the brain liquid (OMS) was checked. Cytokines level, including IL- 1b, IL-1RA, TNFa, IL-6, IL-10 substances, ImmunoChem-2100 analyzer using was evaluated.

Statistics analysis. Statistics for From Statistica 8.0 was used. Indicators medianna, high and lower quartiles with described. Mann-Whitney, Kraskel-Wallis analysis and Spearman correlation coefficients applied. Results considered important (p< 0.05p < 0.05p<0.05).

Results and analysis. Illness start: PD's one bilaterally the beginning it was observed in the majority (84%). Usually disease in hand discomfort or from tremor begins. Disease in 11% of patients one in the leg started, and in 5% two bilaterally common rigidity and hypokinesia with start note done Clinical lateralization and movement disorder weight between dependence not found (p=0.07p = 0.07p=0.07).

Clinical symptoms

Patients between the following clinical symptoms the most many p encountered:

- The intestine on discharge difficulties (85%),
- Fatigue (69%),
- Insomnia (68%),
- Depression (63%),
- Saliva leakage (60%),
- Hyperhidrosis (56%),
- Nocturia (59%).

Cytokine levels.

- IL-1b: in PD patients control to the group relatively significant level high was (p=0.001p = 0.001p=0.001). IL-1β levels in OMS blood to plasma relatively high is 6 (4-7) pg / ml the organize did
- IL-1RA: in PD patients control to the group relatively lower (p=0.002p = 0.002p=0.002). in OMS level 29 (9.5-45.5) pg / ml.

Cytokines level disease stages of symptoms weight and development to the speed looking difference did For example, IL-1b is fast development to the pace have in patients higher is slow to those who develop than significant difference did (p=0.04p = 0.04p=0.04).

Peripheral in the blood interleukins degree research to do neurodegenerative to diseases played in patients long from time since take going, however peripheral indicators based on central nerve system

(MAT) processes about conclusion to do how much right that about question still is relevant. It is known that it is peripheral cytokines in MAT of inflammation development and to increase two road through humor and nerve fibers through, in particular, chest-abdomen nerve fibers through help gives The brain inflammation and peripheral inflammation between dependence confirmation for one row studies the results to bring can One on the other hand, experimental to animals of lipopolysaccharide pyrogen doses peripheral road with when sent nigrostriatal of neurons dead to be and Parkinson similar to disease (PD). clinical appearances development observed. Other from the side, I. Campbell [9] own in his research experimental of animals the brain injection of IL-1P into tissues liver by to inflammation about of cytokines active synthesis cause release showed. From this except, peripheral immune the answer is blood-brain α- synuclein through membrane potential (QMT). aggregates to the blood output as a result the beginning possible it is said [2]. Thus, experimental studies peripheral of interleukins neurodegeneration start and / or strengthen ability, as well as neuroinflammation peripheral the answer to call possible confirms.

MAT inflammation and peripheral immune answer between of dependence clinical evidence as, in the blood high IL-6 level PK development risk to increase, on the contrary, through QMT bad passing nonsteroid to inflammation against drug - flurbiprofen Application PK development risk decrease about information cited [10]. Central and peripheral cytokines in the pathogenesis of PD one different level contribution appendicitis possibility, or some pathogenesis stages peripheral cytokines superiority to do possible guess is being done [11].

The PD duration we determined and IL-6 levels between reverse correlation, T. Muller and by others [12]. take went, only to parkinsonism against therapy didn't get of IL-6 concentration in patients to change note reached data with together with IL-6 production in release the most big change basically PD's initial, perhaps even doclinic stages happened to be possible shows. The level of IL-6 in the blood and PK phase between of dependence lack of while this cytokine of hypergeneration primary feature to show can.

Conclusion. In the pathogenesis of Parkinson's disease (PD). peripheral and central inflammation processes between mutually dependence important place holds Studies that's it shows that peripheral in the blood inflammation mediators, in particular, the level of IL-6, central nerve in the system neuroinflammation and neurodegeneration cause issuer main of the factors is one Peripheral and central cytokines mutually effect of the disease development and symptoms formation contribution adds Especially like IL-6 inflammation of mediators PD's early stages work release disease in development immunological of mechanisms main role shows.

Also clinical observations peripheral inflammation reducing drug means, for example, nosteroid to inflammation against development of drugs, PD risk reduce opportunity confirms. This data, PD early diagnosis and treatment for immunological from markers use prospects determines. With that together, of cytokines degrees and of the disease clinical manifestation to be between dependence more deeper learning necessary.

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