Bulletin of Environment, Pharmacology and Life Sciences
Bull. Env. Pharmacol. Life Sci., Vol 12 [10] September 2023 : 76-79

Online ISSN 2277-1808
Journal's URL:http://www.bepls.com
CODEN: BEPLAD

ORIGINAL ARTICLE **OPEN ACCESS**

Achieving Economic-Effectiveness of Nonpharmacological Interventions In Cognitive Disturbance in Vascular Parkinsonism

Aida Eshmurzaeva, Temur Malik Murotov, Shoh-Jakhon Khamdamov
Tashkent Medical Academy, Republic of Uzbekistan
Tashkent State university of Economic
Email: sh.xamdamov@tsue.uz

ABSTRACT
The article you're referencing seems to discuss advanced treatment strategies for vascular parkinsonism and Parkinson's disease, particularly emphasizing the importance of early detection and treatment of cognitive impairment associated with these conditions. It highlights that elevated serum cortisol levels may serve as an early indicator of cognitive decline, suggesting that timely intervention could prevent significant cognitive deterioration. The article also describes the use of temporal correction, a method of therapy and neurorehabilitation for Parkinson's disease, and reports on its effectiveness.
Key words: *Vascular Parkinsonism, Parkinson's disease, World Health Organization, Alzheimer's disease, Tashkent Medical Academy, Chronic cerebral ischemia, mineralocorticoid receptors, Mini Mental State Examination, Temporythmal correction*

Received 23.06.2023 Revised 13.08.2023 Accepted 21.09.2023

INTRODUCTION

The information you've provided outlines that Vascular Parkinsonism (VP) represents a relatively small percentage of all parkinsonian syndromes, yet it has a significant impact due to its association with various cerebrovascular disorders. The study by Jellinger you mentioned gives insight into the pathomorphological aspects of VP, highlighting the prevalence of cerebrovascular causes, such as multi-infarct scenarios and leukoencephalopathy, and their role in the development of parkinsonian symptoms. It also suggests that a substantial proportion of VP cases may be linked to mild transient forms of the disease and that ischemic strokes and chronic cerebral ischemia can manifest with parkinsonian symptoms, which might indicate early-stage neurodegenerative processes and subcortical structural damage. This underscores the importance of recognizing VP as a distinct clinical entity with both vascular and neurodegenerative components.

MATERIALS AND METHODS

The clinical studies you're referencing provide insight into the etiology of Vascular Parkinsonism (VP), predominantly implicating small vessel disease as a causative factor. Hypertensive microangiopathy, characterized by lipohyalinosis, is frequently identified as the principal cause. This condition leads to diffuse bilateral ischemic damage in the brain's white matter, particularly in the periventricular zone, and is also associated with numerous lacunar infarcts. These vascular changes can result in demyelination, oligodendrocyte death, and axonal dysfunction without necessarily causing outright necrosis. Factors such as hypertension-induced permeability changes in the blood-brain barrier and other arteriopathies, including amyloid microangiopathy and vasculitis, play significant roles in the pathogenesis of VP. Damage to deep brain structures might also arise from hemodynamic disturbances and arterio-arterial embolism due to pathology in larger vessels. Less commonly, midbrain hemorrhages due to arteriovenous malformations can present as VP.

The clinical course of Vascular Parkinsonism (VP) is characterized by several distinctive features when compared to idiopathic Parkinson's disease. VP typically presents with bilateral onset and symmetry of symptoms, less responsiveness to dopaminergic treatment, and a more pronounced impact on the axial parts of the body and legs, leading to early postural instability and gait difficulties. Accompanying symptoms may include signs of pyramid syndrome, cerebellar ataxia, severe pseudobulbar syndrome, frontal lobe symptoms such as the grasp reflex and paratonia, early onset dementia, absence of

hallucinations and delusions, focal neurological deficits like aphasia and apraxia, and early onset of neurogenic urinary disorders. These features differentiate VP from other forms of parkinsonism and guide clinicians in diagnosis and management. Vascular Parkinsonism (VP) can emerge following one or more ischemic strokes or after multiple transient ischemic attacks (TIAs). Extrapyramidal symptoms may manifest acutely or may be delayed, appearing after the regression of pyramidal and cerebellar symptoms. Typically, VP progresses slowly as a result of chronic cerebral ischemia. Recent observations suggest an interplay between cortical functions, particularly in the insular cortex, and serum cortisol levels in the development of parkinsonism, where cortical dopaminergic disruptions might be linked to personality changes and hemispheric symptoms observed in Parkinson's disease. [2].
In addition, due to the high sensitivity of the dopaminergic system, stress increases the development of neurodegenerative processes.

RESULTS AND DISCUSSIONS

Parkinson's disease is considered one of the current topics of today's neurology and geriatrics. Today, the etiology, pathogenesis of this disease and the risk of complications caused by the disease are being thoroughly studied. One of the risks of such complications is cognitive impairment. Today's neurology shows that cognitive disorders are caused by dopamine metabolism disorders, cerebral blood circulation disorders, changes in substance metabolism and a number of other factors. One of the proposed causes and theories is the change in the plasma concentration of the hormone cortisol in Parkinson's disease. Although the role of hormone changes in Parkinson's disease in causing cognitive disorders has not been fully confirmed, this theory has been supported by several years of research by a number of scientists [1].
The study referring to investigates the relationship between cognitive impairment in Parkinson's disease, Vascular Parkinsonism, and other neurodegenerative diseases with elevated cortisol levels. This reflects a broader interest in neurology and geriatrics in understanding the etiology and pathogenesis of Parkinson's disease and its complications. The research methods include clinical examinations conducted over several years in Tashkent, involving patients divided into three groups. While the connection between cortisol levels and cognitive impairment in Parkinson's disease is not yet fully established, ongoing research is delving into this potential correlation. to study the correlation of cognitive impairment in Parkinson's disease, Vascular Parkinsonism and other neurodegenerative diseases with increased levels of cortisol in the blood serum.
Clinical examination was conducted between 2015 and 2022 in the 1st and 2nd neurology departments of Tashkent Medical Academy, 15th and 16th family polyclinics in Olmazor district of Tashkent city. A total of 115 patients participated in the clinical observation. The average age of patients was 56.7 years. Patients were studied in 3 groups.
The clinical research study presented in Table 1 shows characteristics of groups involved in a study on cognitive impairment in Parkinson's disease and its correlation with cortisol levels. There are three distinct groups:
1. **Patients with Parkinson’s Disease**: Comprises 36 individuals, with 12 females and 24 males, having an average age of 62.3±1.1 years.
2. **Patients with Parkinsonism**: Consists of 32 individuals, with 12 females and 20 males, with an average age of 66.7±1.1 years.
3. **Healthy Control Group**: Includes 47 individuals, with 22 females and 25 males, having a significantly lower average age of 34.9±1.3 years.
This table provides demographic data for the study, indicating that the healthy control group is younger on average than the groups with parkinsonian disorders.
In the study, a comprehensive clinical neurological and anamnestic examination following established standards was performed across the three groups. Parkinson's disease severity was measured using the Hen and Yar scale, while Vascular Parkinsonism was assessed with Levin's criteria. The study excluded juvenile Parkinsonism cases. Cortisol levels were measured using an immunofluorescent method, and statistical analysis was performed with nonparametric methods and a significance level set at p=0.05. The data analysis was conducted using the STATISTICA software for Windows 6.0. This structured approach aimed to ensure the reliability and accuracy of the study's findings.
During the clinical follow-up, the results of the concentration of the cortisol hormone in blood serum on the 2nd day of hospitalization in all patients are presented in Table 1

Table 1Periods of incidence of main group PD

	1-5 years	5-10 years	More than 10 years
Male	13(19.1%)	18(26.4%)	8(11.8%)
Female	14(20.5%)	8(11.8%)	7(10.3%)

Table 2: 20 patients with vascular parkinsonism (29%) had a normal level of cortisol hormone, 37 patients (54.4%) had a moderate increase in cortisol, and 11 patients (16.1%) had a high level of cortisol.

Groups	50 – 250 mg / ml	250 – 500 mg / ml	500 - 900 mg / ml
Basic group n=68	20 (29.4%)	37(54.4%)	11(16.1%)
Control group n=47	32(68%)	9(19.1%)	3(6%)

Table 3. **Memory impairment in patients was assessed using the MMSE scale. Mini Mental State Examination MMSE scale score results**

	Below 10 points	In the range of 11-19 points	In the range of 20-23 points	24-27 points	In the range of 28-30 points
Basic group	n=6 8.2 ±2.1	n=19 17.64±4.3	n=16 22.34±2.1	n=18 26.±2.4	n=9 28.67±4.3
%	8.9%	27.94%	23.5%	26.47%	13.23%
Control group	-	n=12 18±2.1	n=22 22.71±5.1	n=6 26.4±1.1	n=18 29±1.1
%	0%	25.53	46.8%	12.7%	38.3%

During the clinical study, the Temporithmic Correction (TRC) neurorehabilitation method was applied to patients to enhance small motor skills. This regimen involved a blend of light physical therapy exercises coupled with medical treatment. Initially supervised by a physician, patients later continued TRC independently at home. Exercises focused on the limbs' distal ends at first, with the exercise duration increasing from the third day. Measurements of step length and height, along with a detailed questionnaire assessing self-care, daily, and social activities, were used to evaluate progress before and after the 10-day TRC intervention.

Table 4 Since the TRC method is used in combination with drug treatment, it reduces the days of drug treatment in VP and PD, reduces the days of doctor's visits and hospitalization.

	The average stride length before TRC is centimeters		The average step length in the 3rd day of TRC is cm		The average step length on day 6 of TRC is cm		The average length of the step on day 10 of TRC is cm	
	Female	Male	Female	Male	Female	Male	Female	Male
Parkinson's disease patients n=36	58.3±1.1	62±1.1	59.2±1.4	63±1.1	61.2±1.2	64.1±1.1	63.2±1.3	66±1.2
Patients with vascular parkinsonism n=32	62.1±1.1	64.1±1.4	63.4±1.3	65.1±1.1	64.3±1.1	66.3±1.1	66.5±1.1	67.5±1.1
Control group n=47	69±1.1	73±1.4	69±1.2	73±1.2	73±1.1	74±1.23	72±1.1	74±1.1

The studies suggest that the prevention of cognitive disorders in Vascular Parkinsonism (VP) and Parkinson's Disease (PD) may be linked to the regulation of serum cortisol levels. The TRC method, combined with the standard treatment, has shown high efficacy in these conditions. While the research supports findings from other studies, it also indicates that the relationship between cortisol levels and cognitive impairment may be more complex, involving factors such as circadian rhythm changes. The study acknowledges limitations, including the use of brief neuropsychological questionnaires, which could affect the conclusions drawn about cognitive functions. Further research with more comprehensive assessments is suggested to refine these findings.

CONCLUSION

The referenced research indicates a correlation between elevated cortisol levels and the onset of cognitive dysfunction in both vascular parkinsonism and Parkinson's disease. Furthermore, the Temporitmal correction method appears to be a highly effective therapeutic approach in managing these conditions, improving gait disorders, and enhancing patients' quality of life with statistical significance ($p \leq 0.05$). This suggests that incorporating TRC into the treatment protocol for VP and PD could be beneficial for patients.

REFERENCES

1. D. Akramova, G. Rakhimbaeva (2018). Hormonal pathogenetic effects and vascular-immunological aspects in secondary vascular parkinsonism, Parkinsonism & Related Disorders,Volume 46, Page e6
2. Hartmann Veldhuisb M. Deuschlea H. Standhardta (1997). Twenty-Four Hour Cortisol Release Profiles in Patients With Alzheimer's and Parkinson's Disease Compared to Normal Controls: Ultradian Secretory Pulsatility and Diurnal Variation, Neurobiology of Aging Volume 18, Issue 3, Pages 285-289
3. K.Phillips, P.Salmon, C.Siwik, W.Rebholz, E.Cash, I. Litvan, J.V.Filoteo, K.Kayser,S.E.Sephton,, A dyadic Mbsr intervention for Parkinson's disease patients/caregivers: Effects on distress, cortisol, and inflammation, Brain, Behavior, and Immunity, Volume 66, Supplement, November 2017, Pages e19-e20
4. David Sancho Cantus, Natalia Santiesteban López, Silvia Solera Gómez et al (2018). Stress in Parkinson's disease. Cortisol and amylase biomarkers. Systematic reviewEl estrés en la enfermedad de Parkinson. Revista Científica de la Sociedad Española de Enfermería Neurológica, Volume 50, Pages 12-22
5. Hemraj B. Dodiya, Christopher B. Forsyth, Robin M. Voigt, Phillip A. Engen, Jinal Patel, Maliha Shaikh, Stefan J. Greenc Ankur Naqib, Avik Roy, Neurobiologyof Disease, 21 December 2018
6. Vladimir S. Kostich, Dusica Lechich, Miroslava Doder, Jelena Marinkovi, Sasa Filipovic, (1996). Prolactin and cortisol responses to fenfluramine in Parkinson's disease Biological Psychiatry Volume 40, Issue 8, Pages 769-775
7. Aleksandar Videnovic, Diego Golombek, (2017). Circadian dysregulation in Parkinson's disease, Neurobiology of Sleep and Circadian Rhythms, Volume 2, Pages 53-58
8. Xiaomei Zhong, Yuping Ning, Zhangying Wu, Cong Ouyan, (2018). A reliable global cognitive decline and cortisol as an associated risk factor for patients with late-life depression in the short term: A 1-year prospective study,Journal of Affective Disorders ; Pages 214-219
9. 9.Qijun Le, Jiabao Hu, Xiaohuan Cao, Siwen Kuang, Man Zhan (2019). Transcriptomic and cortisol analysis reveals differences in stress alleviation by different methods of anesthesia in Crucian carp (Carassius auratus), Fish & Shellfish Immunology , Pages 1170-1179
10. R. Sathiyaa, T. Campbell, M.M. Vijayan (2001). Cortisol modulates HSP90 mRNA expression in primary cultures of trout hepatocytes. Comp. Biochem. Physiol. B Biochem. Mol. Biol., 129; pp. 679-685
11. Robert H. Pietrzak, Simon M. Laws, Yen Ying Lim, Sophie J. Bender(2017). Plasma Cortisol, Brain Amyloid-β, and Cognitive Decline in Preclinical Alzheimer's Disease: A 6-Year Prospective Cohort Study, Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, Pages 45-52
12. Yoshinao Katsu, Taisen Iguchi, in Handbook of Hormones, 2016
13. Namni Goel, ... David F. Dinges, Principles and Practice of Sleep Medicine (Fifth Edition), 2011

CITATION OF THIS ARTICLE

Aida E, Temur M M, Shoh-Jakhon K. Achieving Economic-Effectiveness of Nonpharmacological Interventions in Cognitive Disturbance in Vascular Parkinsonism. Bull. Env. Pharmacol. Life Sci., Vol 12[10] September 2023: 76-79.