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Literature Review

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Alzheimer's disease: is there a way out of the deaf corner?

Abstract

Alzheimer's disease and primary open-angle glaucoma have common links of pathogenesis, so they may have the same trigger - an excessively narrowed pupil, manifested by activation of the parasympathetic nervous system. It is advisable to start the treatment of Alzheimer's disease with medical or surgical dilation of the pupil. This approach can be useful in the treatment of migraine, epilepsy, schizophrenia, post-traumatic stress disorder and other diseases that begin with excessive activation of the parasympathetic nervous system.

Keywords: alzheimer's disease, parasympathetic and sympathetic nervous systems, pupil, mydriatics, laser mydriasis

Volume 14 Issue 5 - 2023

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Received: August 30, 2023 | **Published:** September 14, 2023

Literature review

Alzheimer's disease (AD) is the most common type of dementia and is among the top ten leading causes of death in the world.[1–7] It is considered a multifactorial disease, the trigger of which is unknown. There are no specific drugs.[6,7–15] The purpose of the work: to develop a hypothesis of the development of AD and to propose a new method of its treatment.

Recently, many works have appeared that point to common links in the pathogenesis of HA and primary open-angle glaucoma (POAG) - genetic, vascular.[2–9,14,16–20] POAG ranks second among the causes of blindness in the world, it is considered a multifactorial disease with an unknown trigger, there is no radical treatment.[1,13] Both diseases - AD and POAG - are related to age. In the process of aging, their specific weight increases. Given the common links of pathogenesis, they may have the same trigger.

We offer our view on the etiopathogenesis of these diseases. With age (after 40 years), the pupil progressively narrows. There may be a common trigger in AD and POAG - excessively constricted pupils.[11] It is known that «all diseases are caused by nerves.» In genetically predisposed individuals, chronic stress causes - through cortico-nuclear pathways - excessive narrowing of the pupils. Narrowed pupils (reduction of light flow) trigger pathological biochemical processes in the body through the optic-vegetative system, which leads to deterioration of the mental and physical conditions of patients. Where there is a «weak link» (genetically programmed) in the body, it «breaks» (a pathological process takes place).

Indeed, everything in the human body is interconnected. The eye is an important component of the optic-vegetative system: eye - hypothalamus - pituitary gland. Thanks to the stimulating effect of light in the body, hormones are produced by the glands of internal secretion: pituitary, adrenal, thyroid, sex and others. That is, the eyes provide not only vision, but also the harmonious development of all organs and systems of the body.

If the body - through the cerebral cortex, hypothalamus, reticular formation - affects the size of the pupil, then, according to the principle of «direct connection - feedback», the size of the pupil also affects the entire body. When the pupil is dilated - the mood is elevated, the person is physically and intellectually active; when the pupil is narrowed - the mood, physical and intellectual activity are reduced.[12]

With excessive narrowing of the pupils, the parasympathetic nervous system is significantly activated. An example from practice: a boy, 10 years old, has been under stress for a long time (parents' divorce). The child's mood is depressed. The mother noticed that while working at the computer, her son developed rhinorrhea, sneezing, and coughing.

Chronic stress caused a narrowing of the pupil, and additional miosis at close visual load activates the parasympathetic nervous system, which is realized in the above-mentioned symptoms. It is recommended to limit the use of gadgets, walks in the fresh air, communication with relatives, friends, sports, sufficient sleep, positive emotions (everything that activates the sympathetic nervous system and expands the pupil). It has been established that the activity of cholinergic neurons decreases with AD. And cholinesterase inhibitors (donepezil, galantamine, rivastigmine) are used to treat AD, which reduce the rate of destruction of acetylcholine (a mediator of the parasympathetic nervous system) and contribute to its accumulation in the brain. This therapy is ineffective[6,7,15,16,19] Why?

Let's look at the statistics. In the USA: population group 65-74 years old - 1.6% had AD, 75-84 years - 19% had AD, older than 84 years - 42% had AD.[16] These statistics become understandable if we assume that the trigger of HA is an excessively constricted pupil, the diameter of which progressively decreases with age. In favor of this hypothesis, the fact that the narrowing of the pupil with drops of pilocarpine causes, in addition to eye diseases, exacerbation of AD in susceptible people.[10]

In our opinion, the decrease in the activity of cholinergic neurons registered with AD is a compensatory protective reaction of the organism with AD to the sharp narrowing of the pupil and excessive activation of the parasympathetic system. Apathy, anxiety - early symptoms of AD - occur when the pupil is excessively narrow.[12] The use of cholinesterase inhibitors only worsens the situation.

When the parasympathetic nervous system is overexcited, there is a compensatory activation of the sympathetic nervous system - excessive dilation of the pupil, which is accompanied by excitement, delirium, aggression. Protopopov's triad in the manic phase of bipolar affective disorder is indicative in this regard: mydriasis, tachycardia, constipation (activation of the sympathetic nervous system).

Antipsychotic drugs (adrenoblockers) treat this condition symptomatically, relieving symptoms, but do not affect the trigger of the disease. It is not surprising that they have serious complications (cerebrovascular and motor disorders, reduced cognitive abilities, etc.), which excludes their daily use.

What do we offer?

Dilate the pupil moderately in elderly patients. It is known that in 70% of cases of AD, it is possible to prevent it, which is achieved by physical, intellectual, and social activity.[3] Faith in God, good deeds, professional employment, giving up bad habits, family warmth, travel, and the presence of a goal in life contribute to the expansion of the pupil and the prevention of AD.

In the case of severe concomitant diseases (hypertension, diabetes, obesity, etc.), when various types of activities are difficult, the following ways of solving the problem of AD can be considered:

I. eye drops - mydriatics. Pharmacologists must develop a new class of these drugs that moderately dilate the pupil, are long-acting, and, preferably, minimally affect accommodation. It is advisable to carry out such therapy under the control of biomarkers of the activity of the parasympathetic and sympathetic nervous systems.

II. Dosed surgical dilation of the pupil - laser mydriasis. Operation parameters should be established experimentally.

The proposed approach may be useful for migraines, epilepsy, schizophrenia, post-traumatic stress disorder (which is especially important for countries engaged in hostilities) and other diseases that begin with depressive disorders (ie activation of the parasympathetic nervous system).

So, in our opinion, mental illnesses (like most others) are coded in the size of the pupil. The pupil is the most important structural element of the body, and the Bible spoke about it thousands of years ago: «Take care of me like the pupil of Yours, the daughter of the eye...» (Psalm 17:8).

Thus, the trigger of AD can be an excessively narrowed pupil, and it is necessary to start treatment with its medical or surgical expansion.

Conclusion

Alzheimer's disease and primary open-angle glaucoma have common links of pathogenesis, so they may have the same trigger - an excessively narrowed pupil, manifested by activation of the parasympathetic nervous system. It is advisable to start the treatment of Alzheimer's disease with medical or surgical dilation of the pupil. This approach can be useful in the treatment of migraine, epilepsy, schizophrenia, post-traumatic stress disorder and other diseases that begin with excessive activation of the parasympathetic nervous system.

Acknowledgments

None.

Conflicts of interest

There is no conflicts of interest.

References

1. Allison K, Patel D, Alabi O. Epidemiology of glaucoma: the past, present and predictions for the future. *Cureus*. 2020;12(11):e11686.
2. Chan JW, Chan NCY, Sadun AA. Glaucoma as neurodegeneration in the brain. *Eye and Brain*. 2021;13:21–28.
3. Dementia and Alzheimer's disease: emphasis on the prevention of the progression of cognitive disorders. *Health of Ukraine*. 2021;4:11–13.
4. Gulieva RN. Structural changes of the retina in patients with alzheimer's disease. *Ophthalmological journal*. 2019;4:33–37.
5. Keenan TDL, Goldacre R, Goldacre MJ. Associations between primary open angle glaucoma, Alzheimer's disease and vascular dementia: record linkage study. *Br J Ophthalmol*. 2015;99:524–527.
6. Kuang G, Salowe R, O'Brien J. Genetic factors, Implicated in the investigation of possible connections between alzheimer's disease and primary open angle glaucoma. *Genes*. 2023;14:338.
7. Lukyanets OO. Alzheimer's disease: modern hypotheses of pathogenesis, prospects for the development of the latest methods of early diagnosis and treatment. *Visn Nac Acad Nauk Ukr.* 2021;4: 22–28.
8. Mroczkovska S, Shokr H, Benavente–Perez A, et al. Retinal microvascular dysfunction occurs early and similarly in mild alzheimer's Disease and primary open–angle glaucoma patients. *J Clin Med*. 2022;11:6702.
9. Nation DA, Sweeney MD, Montagne A, et al. Blood brain barrier breakdown in an early biomarker of human cognitive dysfunction. *Nat Med*. 2019;25(2):270–276.
10. Reyes PF. Mental status changes induced by eye drops in dementia of the Alzheimer type. *J Neurol Neurosurg Psychiatry.* 1987;50:113–115.
11. Rudkovska OD. Glaucoma – A new look at etiopathogenesis and treatment. *EC Ophthalmology.* 2022;13(10):26–27.
12. Rudkovska OD. Depression: a new look at etiopathogenesis and treatment. *J Psychol Clin Psychiatry*. 2022;13(3):71–72.
13. Schuster AK, Erb C, Hoffman EM, et al. The diagnosis and treatment of glaucoma. *Dtsch Arzteble Int*. 2020;117:225–234.
14. Sen S, Saxena R, Tripathi M, et al. Neurodegeneration in Alzheimer's disease and glaucoma: overlaps and missing links. *Eye*. 2020;34:1546–1553.
15. Tiwari S, Atluri V, Kaushik A, et al. Alzheimer's disease: pathogenesis, diagnostics, and therapeutics. *International Journal of Nanomedicine.* 2019;14:5541–5554.
16. Trufanov EA. Alzheimer's disease: diagnostic and treatment standards. *Psychiatry psychotherapy and clinical psychology*. 2018;9(4):608–614.
17. Zabel P, Kaluzny J, Wilkose–Debczynska M, et al. Comparison of retinal microvasculature in patients with alzheimer's disease and primary open–angle glaucoma by optical coherence tomography angiography. *Invest Ophthalmol Vis Sci*. 2019;60:3447–3455.
18. Zheng C, Liu S, Zhang X, et al. Shared genetic architecture between the two neurodegenerative diseases: Alzheimer's disease and glaucoma. *Front Aging Neurosci.* 2022;14:880576.
19. Wake R. Long–term effects of combined treatment with memantine and donepezil on alzheimer's disease patients: 72–week study. *Neuropsychiatry*. 2018;8(3):951–960.
20. Xu XH, Zou JY, Geng W, et al. Association between glaucoma and risk of Alzheimer's disease: a systematic review of observational studies. *Acta Ophthalmol*. 2019;97:665–671.

Citation: Rudkovska OD. Alzheimer's disease: is there a way out of the deaf corner? *J Psychol Clin Psychiatry.* 2023;14(5):136–137.
DOI: 10.15406/jpcpy.2023.14.00742