Journal of Alzheimers Disease & Parkinsonism

Drozdova, J Alzheimers Dis Parkinsonism 2021, 11:4
ISSN: 2161-0460

Short Communication Open Access

Alzheimer Disease: Causes, Pathogenesis, Conclusions

Elena Viktorovna Drozdova*

Department of Surgery, Samara State Medical University, Samara, Russia

Description

Alzheimer disease is a severe neurodegenerative disease that is characterized by inevitable progression with loss of social adaptation because of personal and environmental disorientation, end eventually death of a patient.

The Alzheimer disease mortality rate is 37.3 per 100000 populations [1].But the morbidity of this disease has risen significantly in the last 20 years with the average increase of 55.47% through the world [2].

There is some correlation between the level of education and the frequency of the morbidity of dementia.

As the brain is one of the most intensive consumers of energy, we can assume that there is a physiological issue in this pattern of limited education in the background of patients with Alzheimer disease. Thus, in this instance, there is a strong reason to suggest, that this disease is a type of metabolic disease that occurs via chronic subclinical hypoglycemia which in turn causes insufficiency of formation of ATP and other high-energy substances by the neurons and glia. Thus the release of acetylcholine becomes exceeding for the receptors and they block the depolarization that in turn causes the desensitization of the receptors to the acetylcholine.

Hypoglycemia indirectly provokes the decrease of some glucose-associated hormones release (like serotonin), and probably can directly steal the pentose phosphate pathway with the formation of lactate in neurons. The lack of excitability of neurons causes the reduction of metabolism by the accompanying tissue that, consequently, primarily leads to a death of astrocytes and further to a death of neurons. Moreover, the catabolism of Tryptophan ends with synthesizes of kynurenine and its further breakdown products that primarily can stimulate the production of ATP and glycolysis and influence the release of serotonin and may be considered as a compensatory mechanism. But further, the excessive breakdown of Tryptophan probably leads to non-reversible changes in the function of NMDA receptors and causes the direct neurodegenerative changes in a brain. Moreover, occurring via nACh-receptors in the conditions of hypo polarization and in the presence of Mg^{2+} this process additionally can cause the block of the Ca^{2+} current, and consequently exocytose. And knowing that affected by this disease brains often contain Li^{+} ions-additionally highlights the significant role of Mg^{2+} in the blocking of neurotransmissions, because Li possesses similar properties like Mg^{2+}.

According to this theory we can assume that the affected brains contain some elements like Li, Al and so on, that may be the consequence of catabolism of pyrrole, which in some conditions can exhibit the main properties, while β-amyloid plaque and tau-tangles are the trace of a breakdown of the bilipid layer of the membrane of the victim neurons and the remains of a glia. In this case we can explain why the diffuse location of plaques and tangles expresses the most severe brain damage-as it displays the death of mostly interneurons.

But perhaps, the main question is why the morbidity rate of this disease has risen to 55.47% in the last 20 years throughout the world.

None of non-communicable diseases can display such significant growth, without additional, extensive usage of in-depth methods of examination.

I suppose that this is due to an unstoppable passion of people to lose weight using numerous weight- loss drugs, disproportion of proteins and carbohydrates in a daily diet, and probably the smallest proportion of Alzheimer disease is due to an initial proteinopathy-family Alzheimer disease-less than 5%.

That logically contradicts the theory of "excessive" proportion of protein in a daily diet as a cause of dementia (Japan) and that the issue of overwhelming amounts of dementia family diseases is the initial proteinopathy.

Thus, this is the reverse of a diabetes mellitus where the mortality increases from 52 to 380 per 100000 proportionally with the increase of carbohydrates intake in a daily diet from 49% to 74%, regardless of general calories intake. P value=0.001 [3].

A special point I would like to highlight is the WHO statistics that the dementia became 7 of 10 main death causes in 2020 with the significant predominance of women-65%, as an additional evidence of the theory [4-7].

Conclusion

The main reason of this staggering increase of dementia is a wide dieting and usage of weight loss medications with altering both appetite and absorption calories mechanisms.

The proportion of initial proteinopathy within dementia family disease is very low-less than 5%.

The main mechanism of neurodegeneration is chronic hypoglycemia that cause inhibition of glycolysis in glia and block the depolarization of neurons.

The lack of excitability potentiates the synthesis of kynurenine and excessive breakdown of Tryptophan.

The kynurenine and its further breakdown products lead to non-reversible changes in the functions of NMDA receptors and cause the direct and indirect neurodegenerative changes in the brain.

β-amyloid plaque and tau-tangles may be considered as a the trace of a breakdown of the bilipid layer of the membranes of the basic structures of a brain and probably in the presence of the excessive pyrrole breakdown.

***Corresponding author:** Dr. Elena Viktorovna Drozdova, Cardiovascular Surgeon, Department of Surgery, Samara State Medical University, Samara, Russia, E-mail: elenadrozdova.82@mail.ru

Received: April 02, 2021; **Accepted:** April 16, 2021; **Published:** April 23, 2021

Citation: Drozdova EV (2021) Alzheimer Disease: Causes, Pathogenesis, Conclusions. J Alzheimers Dis Parkinsonism 11: 518.

References

1. Statista (2018) Death rate in the United States from 2008 to 2018.
2. Drozdova E (2020) Some controversial nutrition-Associated aspects of decompensation of Oncology and Diabetes Mellitus. Med Clin Res 5: 35.
3. WHO (2019) World Mortality 2019.
4. Alekseevich TO (2020) Biochemistry.
5. Wikipedia (2021) Anti-obesity medications/instructions.
6. Indicator (2021) Vitamin B1 supplementation after spinal cord injury helped improve motor recovery.
7. RayBiotech (2020) Neuroscience Arrays.