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Is Anti-Autoaggressive Treatment Indicated for Dementia?

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ABSTRACT

The incidence of dementia is increasing. To date, there is no promising therapy, nor is there any prophylaxis. The therapy of pathological molecules, e.g. with antibodies, has proven to be insufficient. More recent publications have found that autoaggressive diseases have a dementia-reducing effect. It should be assumed that anti-rheumatic drugs have this effect. Hypotheses are put forward here as to how the pathogenesis of dementia could look so that it reacts positively to anti-autoaggressive agents as an autoaggressive disease. A number of natural remedies are listed that work in this way and are likely to be successful.

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Introduction

There are different theories and ideas regarding the etiology and pathogenesis of dementia. Of course, the pathological proteins such as beta-amyloid and tau protein are known, but their treatment, e.g. with antibodies, has not been able to achieve any significant success to date. It must therefore be assumed that the existing theories are inadequate.

A few months ago, a publication appeared that could provide a new perspective. The team led by Prof. Ali Kiadaliri from Lund University in Sweden conducted a population-based study [1]. The study included 400,000 people who had not been diagnosed with dementia at the start of the study. Those who developed dementia between 2010 and 2019 (N = 22,131) were matched 1:1 with controls for comparison.

Result

People with a diagnosis of arthritis or collagenosis before the start of the study had a lower risk of developing dementia than people without these diseases. The inverse association found between rheumatic diseases and dementia was independent of gender, age and comorbidities.

The fact that rheumatism and collagenosis as diseases could protect against dementia does not appear to be logically explainable. At the end of the study, the authors therefore ask what role the various treatments for rheumatic diseases play in reducing the risk of dementia. This does indeed make sense: anti-autoaggressive treatments (steroidal and non-steroidal anti-inflammatory drugs) could reduce the risk of dementia if dementia were an autoaggressive disease. Other authors have made similar findings [2,3].

An Earlier Publication

The author addressed this problem in an earlier publication [4]. The conclusion was: "We are not powerless in the face of the increase in brain diseases. However, we should take as a pathogenic model the autoaggression within the glia. This results in therapeutic approaches with natural means, which are more promising than the inadequate drug approaches of the industry. With combined applications of these agents, we have seen successes (in prophylaxis and therapy) that exceed what we have seen before. Controlled clinical studies are indicated."The author has recommended naturopathic remedies for therapy: "We have developed a preventive treatment that includes frankincense, myrrh and colloidal gold in a tincture [5]. Other natural remedies that can (or should, resp.) be used successfully here are: Huperzine A, Vinpocetine, Ginkgo biloba, Phosphatidylcholine, Glutathione, Bacopa monnieri N-Palmitoylethanolamide (PEA), Boswellia/ Frankincense and finally Perna canaliculus, the New Zealand green-lipped mussel [4,6-14].

With regard to possible causation, he stated: " The gingival pockets should be cleaned of brain pathogenic and dangerous bacteria such as Porphyromonas gingivalis by rinsing with preparations containing chlorine dioxide [15].

Pathogenesis

How would the progression to dementia be conceivable afterwards, what could the pathogenetic chain look like? If we consider the known pathoproteins as toxins, the process would have to be associated with:

1. toxin accumulation in the brain.
2. detoxification disorder of the brain.

Let's put forward some hypotheses:

- Which toxins can penetrate the brain when the blood-brain barrier is no longer closed, e.g. as a result of exposure to 5G-type technical electrosmog? These are fluorides, light metals such as aluminum and titanium, heavy metals such as mercury, lead, palladium, as well as hydrocarbons of the glyphosate type [16]. Pathogens such as Ebstein-Barr viruses, cytomegaloviruses, HTL viruses, herpes viruses or borrelia (neuro-lyme disease), which cause a smouldering

inflammation, can also penetrate.

- Newer pathogens are the spike(S)glycoproteins (sometimes also called spike proteins) of the SARS-CoV-2 viruses. They attack to ACE2 receptors, in the following order: myocardium, kidneys, brain neurons [17-20]. They cause abnormalities in the cell membranes and their ion channels. The incidence of dementia has increased since the Covid era and the mRNA vaccinations [21].

- What mechanisms can hinder detoxification of the brain? The focus here is on the glymphatic system of the brain, i.e. the lymphatic drainage [22]. It can be impeded by other toxin producers in the head, with the oral cavity being of particular concern, in addition to the paranasal sinuses. Multiple foci may be present here: periodontitis, gingivitis, gangrene granulomas, devitalized tooth roots, metals in amalgam, hardening metals in dental gold (platinum, palladium, etc.), dental electrogalvanic current by different metals, toxins as a result of suboptimally performed dental root treatments, and also "non-infectious chronic jaw ostitis" foci (NICO Neuralgia Inducing Cavitational Osteonecrosis / FDOC = Fatty Degenerative Osteolysis inside the jaw bones) [23]. Of course, any other focus can be involved, e.g. chronic tonsillitis.

Hypotheses

The result of the accumulation of toxins in the glia on the one hand and the lack of detoxification on the other is a silent inflammation, accompanied by a glia-centered mitochondriopathy. This is followed by autoaggression against substances in the plasma up to the cell nuclei of the glial cells, whereby a number of structures and proteins come into question. The glial cells express exosomes for the purpose of detoxification, which cannot be removed but are deposited. The most pathological components of the exosomes are the well-known beta-amyloids and tau proteins. They are at the end of the pathogenetic chain. Their treatment without taking into account the previous links in the chain is inadequate.

Conclusion

From this pathogenetic derivation - as a hypothesis - it follows that anti-autoaggressive agents and medications should be the treatment of choice for dementia. These can be steroidal and non-steroidal anti-inflammatory drugs, but also naturopathic remedies, which have a corticoid-like effect and can slow down the immune system in its inadequate hyperactivity. The best known of these is certainly frankincense, which we have combined with myrrh and colloidal gold [5]. New Zealand green-lipped m ussel has an even stronger effect [24-27]. These remedies can of course also be combined.

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