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Case Report

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Multiple sclerosis: progress in detection and therapy?

Manfred Doepp*

HolisticCenter, 13 Haupt St., Abtwil 9030, Switzerland.

*Corresponding Author
Manfred Doepp, HolisticCenter, 13 Haupt St., Abtwil 9030, Switzerland.

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Abstract

Diagnosis and therapy of multiple sclerosis (MS) are not optimal so far, they need improvement. We present a diagnostic parameter and two therapeutic methods that offer a prospect of success without burdening the patient. They are the biomarker Nf-L for the evaluation of the status of the lesions of the neural axons and the application of two treatment methods of endogenous origin: PEA (N-Palmitoylethanolamin) and intracellular Enzymes. They are suitable for MS in terms of their effects. The good experience gained so far suggests that it would be worthwhile to test them in detailed studies.

Keywords: multiple sclerosis, MRI, Neurofilament Light Polypeptide, Immunoassays, Alzheimer's Disease

Introduction

The incidence of multiple sclerosis (MS) is increasing. Currently, the incidence in Switzerland is about 20,000 cases per 8 million inhabitants. Diagnosis remains difficult unless an MRI is performed in all suspected cases. It would be better, however, if there were a screening method that required only a blood draw. More recently, a laboratory value has emerged for this purpose; it is the biomarker Nf-L. It has great significance for neural axon lesions, so let's take a closer look at it [1].

Nf-L as Diagnostic Tool

Neurofilament light polypeptide, also known as neurofilament light chain, abbreviated to NF-L or Nfl and with the HGNC name NEFL is a member of the intermediate filament protein family. There are four major neurofilament subunits, NF-L, NF-M, NF-H and α-internexin. These form heteropolymers which assemble to produce 10nm neurofilaments which are only expressed in neurons where they are major structural proteins, particularly concentrated in large projection axons.Axons are particularly sensitive to mechanical and metabolic compromise and as a result axonal degeneration is a significant problem in many neurological disorders. The detection of neurofilament subunits in CSF and blood has therefore become widely used as a biomarker of ongoing axonal compromise. The NF-L protein is encoded by the NEFL gene [2,3]. Neurofilament light chain is a biomarker that can be measured with immunoassays in cerebrospinal fluid and plasma and reflects axonal damage in a wide variety of neurological disorders [4,5]. It is a useful marker for disease monitoring in amyotrophic lateral sclerosis [6], multiple sclerosis [7], Alzheimer's disease [8,9], and more recently Huntington's disease [10]. It is also promising marker for follow-up of patients with brain tumors [11]. Higher levels of blood or CSF NF-L have been associated with increased mortality, as would be expected as release of this protein reflects ongoing axonal loss [12].

There is widespread agreement that it is an autoimmune disease [13]. However, the trigger is unclear; among others, the Epstein-Barr virus is suspected [14]. This virus is widespread in the population. It is striking that the further someone lives from the equator, the higher the probability of MS. Perhaps sun exposure, and thus vitamin D3 levels, has a role as a protective factor. Each case of the disease is individual, as is the course: from blande to relapsing to progressive. Therapy is consistently immunosuppressive and thus quite schematic. However, the marker Nf-L has the ability to reflect the activity of the immune process. If the values remain constant or decrease, the therapy can be reduced. One could even postulate that the Nf-L value should be part of a screening program if there is even the slightest indication of a lesion of the axons.

Therapies

As mentioned, immunosuppressants are a standard therapy in MS. By their very nature, they have the disadvantage of increasing the risk of infection. It would be desirable to have agents with fewer side effects.

PEA

The author has had good experience in recent years with the endogenous substance N-Palmitoylethanolamin (PEA) [15]. It is an endocannabinoid with antioxidant effects. The endocannabinoid system (ECS) is a part of the nervous system and includes the cannabinoid receptors CB1 and CB2 with their natural ligands and the downstream intracellular signal transduction after ligand binding in vertebrates. PEA occurs in the stratum granulosum of the skin and possesses, among other things, antioxidant protective effects against UVB radiation.

Cannabinoid receptor 1 (CB1) is found predominantly in nerve cells. It is most abundant in the cerebellum, basal ganglia, and hippocampus. However, it is also found in the peripheral nervous system (e.g. in the intestine).

Cannabinoid receptor 2 (CB2), on the other hand, is found predominantly on cells of the immune system.

Furthermore, the G protein-coupled receptors GPR18, GPR119, and GPR55 are also thought to be cannabinoid receptors in the endocannabinoid system [16-20].

The CB2 receptor is thought to play an important role in the regulation or modulation of the immune system. Since the brain regions where the CB1 receptor is predominantly found play an important role in memory (hippocampus and cerebellum) as well as movement regulation (basal ganglia and cerebellum), it is reasonable to assume that endocannabinoids influence learning and movement processes [21,22]. Other physiological processes involving the endocannabinoid system include pain states, sleep induction, appetite and motility control, temperature control, neuroprotection, and cancer. Endocannabinoids directly inhibit P-type calcium channels in these cells.

Studies [23,24] have been conducted in patients with

- Movement disorders, such as dystonia, Gilles de la Tourette syndrome, Huntington's disease, and
- Parkinson's disease
- Multiple sclerosis, to influence ataxia, neurogenic bladder emptying disorder, pain, spasticity,
- tremor and inhibition of neurodegeneration
- Other diseases associated with spasticity (paraplegia, AIDS encephalomyelopathy)
- Various neurological pain syndromes (various forms of headaches, neuralgias, neuropathies
- Craniocerebral trauma, neurodegenerative diseases, amyotrophic lateral sclerosis (for
- neuroprotection)

PEA is an endogenous substance that acts as a signal molecule throughout the body. The preparation we use (Dologon® from www.drhittich.com) works in synergy with the highly bioavailable BCM-95 Forte® full-spectrum Curcumin (alpha-turmerone and ar-turmerone from the essential oil) and the Xanthin360® full-spectrum Astaxanthin. Astaxanthin is extracted from its best source, the freshwater algae Haematoccocus pluvialis, using the particularly gentle CO2 extraction process. This PEA preparation is 1.8 times better absorbed and utilized than usually available PEA.1 The dose ranges from 400 to 800 mg/day. Immunosuppressants were only required in progressive cases. Up to now we have seen no side effects.

Intracellular Enzymes

In 2019-2021, a group of authors studied how the effect of intracellular enzymes may be on MS [25]. Intracellular enzymes should not be confused with the commonly used digestive enzymes, it is a different class. They are produced only by the Italian company Citozeatec (www.citozeatecsrl.ch). In the manufacturing process, the intermediate intracellular metabolism is imitated. There are twelve different agents for different purposes. They are considered as dietary supplements. The study article: Dietary Supplements in Combination with Conventional Medicine among People with Multiple Sclerosis.

Description of the Biodynamic Enzyme Therapy [26]

First it must be emphazised that without enzymes there is no life. The enzyme means originate from applied biochemistry, i.e. functional nutrition with the help of dietary supplements [27]. Using the latest biotechnologies, Citozeatec produces, through successive enzymatic conversions, agents to promote the energy and metabolic processes of the cells. These can help prevent or counteract chronic degenerative processes. They increase the energy efficiency of cells by reactivating, regulating and enhancing enzyme activity.

In this way, the self-healing powers and regulatory capacity in the organism can be decisively improved.The dietary supplements can be successfully used both preventively and therapeutically. So far, positive results have been achieved in the treatment of various diseases, such as inflammation, auto-immune diseases, viral diseases and tumor diseases.

Biodynamic enzyme therapy sees diseases as expressions of self-regulation. They can be turning points in the gradual process of degeneration and aging. In "Living Food," author Ernst Günter defines enzymes as special substances that contain the spark of life by controlling the biochemical processes of all organs in humans, animals and plants so precisely that they appear to be endowed with intelligence or consciousness.

The knowledge of molecular, biochemical and enzymatic mechanisms in all cells as well as new therapeutic approaches in many pathological processes is of great importance to treat patients successfully and without side effects. Although enzyme therapy has hardly been applied in orthodox medicine to date, it is nevertheless based on the classical medical foundation of biochemical science. This is put here in a unique form "from the head to the feet".

Intracellular enzymes are thus a natural or nature-identical possibility of treatment, also in MS. We have achieved significant treatment successes-also combined with PEA. It is suggested that further scientific research and studies should be done with it.

Conclusion

If the diagnosis and therapy of a disease are not sufficiently successful, one should look for new ways. We present a diagnostic biomarker (Nf-L) that is directly correlated with axonal lesions in MS (and other axonal-destructive diseases). This parameter allows not only diagnosis but also control of therapeutic intensity.

Although its specificity is reduced, any patient with an elevated value must be extensively searched for the cause. For therapy we present two methods, which are organism-identical and in principle have no side effects. It is the substance N-palmitoylethanolamin (PEA) and the substance class of intracellular enzymes. This opens up new possibilities for the therapist.

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