Possibilities of homeopathy in the treatment of demyelinating diseases of the nervous systems

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The article presents some modern concepts of the pathogenesis of multiple sclerosis and discusses the possibilities of using homeopathic medicines for the treatment of this category of patients. The author cites one case of successful homeopathic treatment of multiple sclerosis, which is confirmed by the positive dynamics of MRI studies.

Abstracts: The paper presents certain modern conceptions of pathogenesis of the multiply sclerosis and demonstrates applications of some homeopathic drugs for curing of this pathology. The author gives one case of successful homeopathic treatment of multiply sclerosis, confirmed by repeated MRI-examinations.

Demyelinating diseases, among which multiple sclerosis (SD) is the most common, are, perhaps, the second most severe and disabled patients in the daily practice of a neurologist. On the other hand, this group of pathological processes, with all the ambiguity and contradictory information about the mechanisms of pathogenesis, is a clear example of the helplessness (in practical terms) of the modern molecular biological view of the development of diseases.

It should be noted that after Charcot in the middle of the 19th century described the clinical and pathomorphological manifestations of SD and singled out the disease as a separate nosological unit, this nosology constantly continues to pose questions to neurologists, the answers to which, of course, were and are being questioned.

Opinion about the main etiological factors demyelinating diseases have fluctuated between endogenous and exogenous for more than a century (DS Comston, 1990). A possible genetic predisposition in the genesis of SD has been known for a long time; this idea was first expressed more than a century ago by Strumpel. Recent studies using the twin method (AD Sadovnick et al., 1993) have shown that the role of heredity is large, but in itself insufficient for the development of most cases of SD.The phenotype of the disease has wide variability and suggests an autoimmune nature (G. Ebers, 2002) ...

PATHOGENESIS SD

It is generally accepted that the primary systemic impetus in the development of the disease is carried out by the antigen (viral infection "superantigen"). Once in the peripheral blood, it is phagocytosed by macrophages, which present it

on its surface as part of the receptors of the main histocompatibility complex. During this initial phase, the antigen is recognized by T-helpers with the reactivation of CO4-lymphocytes, including myelin-reactive ones, which are classified as Th1-like.

Antigens in the main histocompatibility complex recognize CD8, the main function of which, as you know, is to inhibit the autoimmune process in its initial phases. Due to systemic immunological dysfunction, this process is disrupted with the transformation of CD8 suppressor inducers into cytotoxic CD8 suppressor effectors. They, in turn, stimulate autoreactive Th1 cells with the subsequent triggering of the autoimmune process by the production of proinflammatory cytokines: IFN-g, LT, TNF-a.

Cascade of these transformations leads To damage Blood-brain barrier (BBB) and autoreactive T-lymphocytes with CD4 phenotype to antigen - myelin basic protein (MBP), proteolipid protein (PLP) or myelin-oligo-dendrocytic glycoprotein (MOG) - penetrate into the central nervous system. It is believed that autoreactive CD4 lymphocytes are the trigger for demyelination in the central nervous system. In the brain tissue, they are reactivated by cytotoxic T-lymphocytes. B-lymphocytes, glial cells and macrophages and trigger a cascade of immune responses: the production of pro-inflammatory cytokines, autoantibodies, proteinases, chemokines, free radicals, NO. Immunological and pathochemical disorders cause the formation of diffuse perivascular foci of inflammation, primarily around postcapillary venous structures, Focuses of inflammation are transformed into active foci of demyelination and axonal damage,

With a favorable course of the disease cell-mediated inflammation can subside under the influence of regulatory T-lymphocytes, anti-inflammatory cytokines - IL-10, IL-4, transforming growth factor - (TGF- (b)), prostaglandin E, which promotes remyelination of damaged axons.

Thus, according to the given pathophysiological concept, the following interrelated processes are the main links of SD pathogenesis:

1. Induction by one or more exogenous agents autoimmune response to myelin proteins.
2. Systemic immune dysfunction initiated by trigger cells Th1 with CD4 phenotype.

Damage to the BBB with increased permeability or dysfunction of the central nervous system.

Major sources of damage:

- inflammatory mediators (cytokines, nitroxide), activated macrophages, autoantibodies;
- Th1 lymphocytes, which enhance the effect of provoking factors;
- Th1 lymphocytes that synthesize autoantibodies.

Stages of brain tissue damage:
• perivascular inflammation;
• demyelination;
• demyelination / remyelination;
• axonal degeneration;
• formation of gliosis (scarring).Despite significant progress in the understanding of molecular biochemical aspects of SD pathogenesis, the exact mechanism of its development is unclear. This is a very difficult task, since many pathological processes occur simultaneously with SD: edema, inflammation, BBB disturbance, de- and remyelination, axonal damage, gliosis; the sequence of their occurrence is not completely known. A variety of pathogenetic models of SD are allowed (C, F. Lucchinetti et al., 1996).

Landry's syndrome is a very formidable acute demyelinating disease that requires urgent resuscitation. Ascending paralysis, first described in 1859 by the French physician Landry, is a symptom complex, which can be based on a variety of etiological factors. Currently, there is a general opinion that the clinical picture, called Landry's palsy, is etiologically very complex, and nosologically does not represent any unity. In most cases, acute infections are the leading etiological factor of this disease. A clinical picture similar to Landry's syndrome is observed in some forms of poliomyelitis, encephalomyelitis, which develops after anti-rabies vaccinations, and paralysis as a result of severe poisoning.

Usually, the disease begins suddenly, often against the background of complete health, and very quickly (in the period from tens of minutes to several hours) the pathological process spreads widely throughout the nervous system, affecting in some cases mainly peripheral, and in others - its central parts. Pregnancy is often the trigger for the development of this acute condition. In this case, the development of this pathology is an absolute indication for immediate medical termination of pregnancy with subsequent sterilization.

The results of pathological studies of polyneuritic forms of the disease showed that in the spinal cord, degenerative changes in the anterior horns, disintegration of myelin in nerve fibers, as well as various changes in the axial cylinders, up to their death, are observed.

According to the literature, before 1960, mortality in these forms was high and amounted to 97%, which was associated with the absence of mechanical ventilation devices. The survivors, with rare exceptions, remained disabled. In recent years, a decrease in mortality has been noted under the condition of modern hospitalization of the patient in the intensive care unit and adequate pulmonary resuscitation.

The clinical picture of the process most often, according to numerous observations,

corresponds to action Conium: feeling tired, muscle relaxation,staggering gait, knees buckling; vision becomes unclear, diplopia; consciousness remains calm and clear, sensitivity is preserved; impaired ability to move; followed by a period of collapse, weakness of the lower extremities, making the gait swaying; muscle strength in the arms is gradually lost, and soon any possibility of voluntary movements disappears; pupils are dilated, motionless, visual disturbance is observed (J. Charette, 1990). A beautiful and efficient use caseConium in intensive care for treatmentthis pathology is described by colleagues from Odessa L.P. Chernobrova and M.P. Stoyanov [6; 7].

It should be remembered that the process of "maturation" of the myelin sheath occurs in the postnatal period under the influence of taurine contained in breast milk. At the same time, attempts are known in allopathic practice to treat SD with bovine colostrum. For us, this is a good clue that makes us think about the use of "milk" preparations (including potentiated colostrum -Colostrum) with this pathology.

Separately, it should be said about the dialectical relationship known in immunology between autoimmune processes and malignant neoplasms. From the point of view of the phasing of the processes, as well as according to the theory of parabiosis N.E. Vvedensky, demyelinating (autoimmune) process and malignant neoplasm are two opposite phases of the immune system's reactions, its hyper- and hypofunction. That is why, according to immunologists, the key to solving these two huge problems of medicine is the same. In homeopathy, this dualism is clearly demonstrated to us by the pathogenesis of some "snake" drugs, in particular,Crotalus horridus, having in its pathogenesisboth destructive oncological processes and multiple sclerosis, ALS, progressive muscular dystrophies. A similar dualism demonstrates to us the analysis of this pathology from the point of view of the theory of miasms. From the course of phthisiology it is known that patients with various forms of tuberculosis (or simply persons who are usually called tuberculosis) very rarely get sick with malignant neoplasms. At one time, this even gave rise to a number of studies on the isolation of substances exhibiting antitumor activity from the culture of tuberculous mycobacteria. Everyday homeopathic practice and case analysis show us that the drugs most often and effectively used in the treatment of demyelinating processes are most often drugs of the psorotuberculin series, and in the family history of such patients, oncopathology, according to our observations,

The observation made by the American physician Betty Martini is of undoubted interest for homeopathy. According to her, excessive consumption of sweeteners (aspartame) leads to the development of symptoms reminiscent of multiple sclerosis and systemic lupus erythematosus. The mechanism of development of this symptomatology, according to research results, is associated with the fact that when heated above 30 ° C, wood alcohol (methanol) contained in aspartame turns into formaldehyde, from which formic acid is then formed, which has a neurotoxic effect.

Considering the above and knowing the pathogenesis Formica rufa, we see that it isthe drug has lower paraplegia, right-sided hemisyndrome, spinal cord injury. If we consider that an excessive desire for sugar is often closely related to alcohol dependence (in Western literature there is even a special term "sugar drinker" by analogy with an alcoholic), then you should think about the possibility of usingFormica rufa and Saccharum officinale not only withdemyelinating pathology, but also with toxic (alcoholic) polyneuropathies, phenylketonuria.

Also, according to recent studies, from the venom of marine invertebrates, you can get an effective cure for multiple sclerosis. Scientists from the University of California at Ervine (Irvine) and the University of Marseilles (Marseilles) managed to create and test a prototype of a drug for this severe neurological disorder. For this, the poison of anemones was used.Stichodactyla helianthus.

In the course of the study, one of the components of this poison, called ShK, was tested in an experimental model of multiple sclerosis. For the simulation, activated cells of the immune system were used, which copied cells that appear in sick people.

These cells, T-lymphocytes, had an unusually large number of special ion channels on their surface. It is these changes that cause the cells of the immune system to attack their own nerve fibers, leading to the characteristic signs of multiple sclerosis, including paralysis and death.

During the experiment, ShK blocked "extra" ion channels, and only one type of channels. This means that the sheath of nerve fibers can be protected from the attack of the immune system, preventing the destructive effect. Now it is necessary to confirm the effect of anemones venom on animals and humans,

According to one of the study's authors, Heike Wulff, although ShK seems to be an effective remedy, it is difficult to use for long-term treatment due to its rapid destruction in the body. Scientists are now trying to find a substance with a similar structure that persists for a long time in the body.

For homeopathy, such a message is of undoubted interest and encourages us to take another closer look at our "sea" medicines (Asterias rubens, Medusa) in terms of the prospects for their use for treatmentthis formidable pathology.

According to the literature and his own observations, when treating demyelinating pathology (SD, Landry's syndrome, ALS, syringomyelia), a homeopathic physician most often has to think about the use of drugs such as Ac. oohlicum, Ac. succinicum(one of the main links of the cell respiration cycle - Krebs tricarboxylic acids, the strongest myelin protector),Agaricus, Alumina, Argentum, Cadmium sult., Crotalus horridus, Hypericum(syringomyelia), Iridium metallicum, (differentiate from Conium maculatum),Lathyrussativus, Oleander, Onosmodium virginianum, Oxytropislamberti, Petroleum, Phosphorus (and phosphorus-containing drugs),Plumbum, Stannum, Thallium, Theridion curassavicum, Zincum (especially Zincum chromicum - spinal

demyelinating processes, syringomyelia).

As rightly noted by many authors, due to the fact that a patient with a demyelinating process gets an appointment with a homeopath, as a rule, after long-term allopathic treatment, often with the use of corticosteroids. blurred clinical picture often complicates the choice of similium; in this case, in our opinion, it is advisable to start treatment with the appointment of a single doseSulfur 10M (with possible gradual withdrawal, hormonaldrugs), which allows you to "clear" the case. In conclusion, let us consider the case of successful homeopathic treatment of a patient with cerebellar-hemispheric form of disseminated encephalomyelitis. confirmed by the data of repeated MRI studies.

CASE 1

Patient Lyudmila B., born in 1953, was admitted with a referral diagnosis of acute cerebrovascular accident to the neurological department of the City Clinical Hospital No. 63 g, Moscow on 15.02.1991. Complaints: general weakness, severe weakness of the right extremities, recurrent headaches, mainly in the left superciliary region, recurrent cramps in the left eye, accompanied by a feeling of "shroud" in front of him.

Anamnesis: sick with 1988, when she began to notice periodically arising systemic dizziness, disorientation, unsteadiness when walking, deterioration of lateral vision in the left eye. At the initial hospitalization in June 1988 (City Clinical Hospital No. 81), a partial paresis of the gaze to the right, central paresis of the VII, XII pairs of cranial nerves on the right was revealed. Was discharged with improvement, with a diagnosis of "vascular crisis". In July of the same year, followed by repeated hospitalization in the clinic of the Central Research Institute of Occupational Hygiene and Occupational Diseases with similar complaints; when examined by an ophthalmologist, a homonymous quadrant hemianopsia was revealed. She was discharged with improvement, with the previous diagnosis; She was re-hospitalized in September of the same year at the Joint Hospital IV of the Ministry of Health of the RSFSR, where a brain computed tomography revealed a focus of low density in the right parietal-occipital region, measuring 3x1.5x3cm. She was consulted by a neurosurgeon with the recommendation of a consultation at the Central Research Institute of Neurology of the Academy of Medical Sciences. Discharged with the previous diagnosis; 10/13/1988 consulted by prof, A, M. Wayne, who was the first to suggest a demyelinating disease.

02/14/1991, while on vacation in Sochi, suddenly developed a weakness in the right extremities, examined by a neurologist and (by air ambulance) hospitalized in City Clinical Hospital No. 63.

Life history: in childhood - frequent colds (with rapiddevelopment of bronchial pathology), nosebleeds, bleeding gums, fear of the dark. On the maternal side - heavy menstruation, on the father side - IHD,

Modalities: thermophilic, prefers cold drinks,loves salty and spicy food, suffers from high hyperthermia, drinks often and a little during fever, loves calm music.

On examination: satisfactory condition, without convincing organicfeatures; BP = 110/70 mm Hg; thin, there is a slight stoop.

In neurological status:conscious, contact, adequate, oriented, cerebral and meningeal symptoms are absent; mild left-sided facial hypoesthesia, mainly in the innervation zone of the first branch of the V pair of cranial nerves, bilateral hypesthesia of Zelder's outer zones, vivid corneal reflexes, congenital left-sided teeth asymmetry; easy installation nystagmus to the left, aggravated by sensitized tests; pharyngeal reflexes are symmetrically somewhat reduced; slight deviation of the tip of the tongue to the right; abdominal reflexes are absent; coarse right-sided flaccid hemiparesis up to 0-1 points in the arm and leg, with increased reflexes, expansion of reflexogenic zones; protective foot reflexes on both sides.

Based on the data of the anamnesis and the clinic, the diagnosis of disseminated encephalomyelitis of the cerebellar hemispheric form was made for the first time. The patient underwent the first course of acupuncture (IRT) according to the Tenk-Werner technique; was discharged with full restoration of the strength of the right extremities, but with the preservation of a decrease in tone in them, with a slight anisoreflexia; abdominal reflexes are absent. At discharge, the setting nystagmus to the left was preserved, slight instability in the Romberg position, which intensified with a sensitized test to the left, performed coordination tests slightly worse on the right. The patient is diagnosed with the 1st disability group.

After discharge, she was consulted in the Department of Slow Infections of the Central nervous system of the Central Research Institute of Neurology of the Academy of Medical Sciences with confirmation of the diagnosis.

Later, in 1992-1998, she repeatedly took outpatient and inpatient treatment courses, repeated courses of IRT (Tenk-Werner method) and dalargin microelectrophoresis (Kondratova-Nechushkin method) were carried out, with a good effect. Dynamic observation using the "SVT CITO" technique showed a significant improvement in vegetative parameters and a decrease in the severity of demyelination; MRI over time showed a decrease in the focus of demyelination to 2x2 cm. With repeated annual re-examination, first the 2nd and then the 3rd group of disability was established.

In April 1999, the patient was assigned Phosphorus C200. on 3 globules at night, 5 doses daily, then 3 globules 2 times a week, for two months. After 1 dose of the drug at night, she noted an ascending numbness of the upper and lower extremities, which regressed within several hours, which frightened the patient very much, since, according to her, it was from these sensations that the disease began in 1988. Upon repeated examination, a complete regression of nystagmus was noted , coordination disorders and sensory disorders in Zelder's zones, discomfort from the left eye; there was a slight decrease in tone and an increase in reflexes in the right extremities, without expansion of reflexogenic zones, abdominal reflexes were still absent. Repeated MRI examination in May revealed no demyelination focus. During the next examination by VTEK in June 1999, the disability was removed.

LITERATURE

1. Zavalishin I.A., Zakharova M.N., Zhuchenko T.D., Peresedova A.V. Etiology and pathogenesis of multiple sclerosis // Multiple sclerosis: selected issues of theory and practice / Ed. I.A. Zavalishin, V.I. Golovkin. - M., 2000.

2. Gusev E.I., Demina T.L., Boyko A.N. Multiple sclerosis. - M.,1997.

3. Kent J.T. Lectures on homeopathicMateria medica. - M., 1999. - T.2.

4. Fatak S.R. A Brief Repertory and Materia Medica of Homeopathic medicines. - Novosibirsk, 2000.

5. Khaidarov B.T. Pathogenetic mechanisms of multiple sclerosis and questions of immunocorrective therapy (clinical, immunological and biochemical research): Dis .... Dr. med. sciences. - M., 1998.

6. Chernobrova L.P., Stoyanov M.P. Homeopathic treatment of the patient ascending paralysis - Landry's syndrome // Homeopathy and herbal medicine. - 1997. - No. 2.

7. They are. Homeopathic treatment of Landry's syndrome // Ukrainian homeopathic schoor! chnik. - Odessa, 1998 .-- T. 1. - S. 100-101.

eight. Paty DW, Li DKB MRI Study Group et al. Interferon beta-1 b is effective relapsing-remitting MS. II MRI analysis, results of multicenter, randomized, double-blind, placebo-controlled trial // Neurology. - 1993. - Vol. 43. - P. 662-667.

nine. PRISMS Randomized double-blind placebo-controlled study of interferon b-1a in relapsing / remitting multiply sclerosis // Lancet. - 1998. -Vol. 352. - P. 1498-1504.

ten. Rudick R., Zoodkin D. Jacobs Let al. Impact of interferon beta-1 a on neurologic disability in replasing multiply sclerosis // Neurology. - 1997. -Vol. 49. - P. 358-363.

eleven. Sorensen PS, Wanscher B., Jensen CV et al. Intravenous immunoglobulin G reduces MRI activity in replasing multiply sclerosis // Neurology. - 1998. - Vol. 50. - P. 1273-1281.

Nechushkin, D.A. Possibilities of homeopathy in the treatment of demyelinating diseases of the nervous system / D.A. Nechushkin // Traditional Medicine. - 2004. - No. 2 (3). - P.34-38.

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