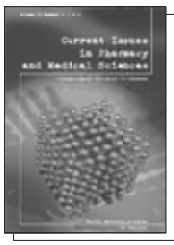

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Multiple sclerosis – causes, risk factors and symptoms of the disease – a literature review

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ABSTRACT

Multiple sclerosis (SM) is a classified chronic disease which leads to disability. Apart from severe cases, persons suffering from SM, live on average, as long as healthy people, however, their everyday life is often considerably difficult. SM usually occurs in young people. The first relapse normally happens between the age of 20 and 40. As a result of the pathologic process, myelin, i.e. the substance from which neuron sheaths are built, is damaged. The process disrupts normal impulse transmission along neural paths in the encephalon and the spinal cord. The causes of SM are unknown, despite broad knowledge of the mechanisms of impairments that occur in the course of the disease. The symptoms of SM can be transient and sometimes seem difficult to describe.

Keywords: multiple sclerosis, causes, risk factors, symptoms

INTRODUCTION

Multiple sclerosis (sclerosis multiplex, SM) is a chronic inflammatory and demyelinatory disease of the central nervous system with an auto-immune background [5,7]. SM is a disease concerning neurons, glial cells (oligodendrocytes) and immune cells of the brain (microglia), in which the myelin sheath is damaged around the neuron bundles. This damage does not allow normal impulse transmission along the neural paths in the encephalon and the spinal cord. The disease is characterised by relapses and remissions, i.e. periods when no symptoms occur. The myelin is dissolved and replaced by scars, i.e. scleroses; damage can occur in various parts of the nervous system and present itself with multiple symptoms, which explains the second part of the term [1].

Presentations of SM. In terms of the clinical course of the disease, the following presentations of SM can be distinguished:

1. The relapsing-remitting sclerosis occurs when new neurological symptoms begin (the so-called relapse) lasting above 24 hours, when at the same time, no other pathological causes which could have triggered the symptoms, are recognised. After some time, there is an improvement and withdrawal of the symptoms, i.e. remission, characterised by the lack of progression of the disease. This is the most frequent presentation of the disease.
2. The primary progressive presentation, where from the beginning, there are no relapses in the pathological process, but the clinical condition continuously declines. The rate of accretion of the neurological deficit is individual for each patient.
3. The secondary progressive presentation is a resultant of the two above-mentioned ones. In this case, after the relapse stage, there is a slow, constant deterioration of the neurological deficit.
4. The progressing-relapsing presentation characterising itself with progression of the disease from the beginning, with relapses. Periods between relapses are characterised with an evident progression of symptoms [5,7].

Causes and risk factors of multiple sclerosis. The causes of multiple sclerosis are not known, despite broad knowledge of the mechanisms of impairment that occur in its course. According to a strictly immunological-based theory, the pathogenesis of SM is explainable in terms of initiating the inflammatory process. In this, T lymphocytes play the main role, passing the blood-brain barrier and entering the central nervous system. In patients with SM, regulatory T lymphocytes are much less capable of inhibiting the division of lymphocytes that recognise myelin as a foreign substance and create a response to it. The ensuing inflammatory process also involves other cells of the immune system, as well as cytokines and antibodies [5]. Despite the fact that the ethiology of the disease has not been recognised fully, factors triggering the disease have been de-

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fined. Endogenic factors are readily apparent, including, *inter alia*, genetically-conditioned predispositions. Furthermore, while SM is not regarded as a hereditary disease, there is some evidence that genetic factors have a share in the predisposition for contracting the disease [1]. Moreover, there is a convergence between SM and some antigens of the HLA system, e.g. HLA3, B7, DR2, DQW1, and a connection between the disease and other gens, e.g., coding proteins of T lymphocyte receptors [5,9].

The endogenic factors also include a disturbed immunological reaction (an immunological defect), abnormal physical and chemical condition of the myelin, as well as disorders in its transformation [1]. In turn, extrinsic factors are infections with a specific virus (retrovirus) or epidemic non-specific virus (para-influenza, measles) where similarity can occur between the antigen of the virus and the antigen of the central nervous system cells. This, in turn, causes the immune system to create a response against its own cells [3,5,9].

In this group, the influence of geographic factors and climate is taken into account; the disease occurs more often in areas located in the moderate climatic zones (Europe, northern parts of the US, southern Canada, southern Australia and New Zealand) [1,2,5]. Extrinsic factors also include nutrition. Recent findings indicate that Vitamin D3 is an important regulator of the immune system and has immunomodulatory properties [2].

The connection between Vitamin D3 deficiency and the risk of SM is consistent with the observed geographic distribution of the disease due to decreased exposure to sunlight. The areas of Alaska and Greenland constitute an exception, i.e. areas with low insolation, where the incidence of the disease is low. One of the hypotheses explaining the phenomenon is a change in incidence connected with a diet rich in ocean fish [9].

A significant connection was also noticed between stress and the deterioration of functioning or the incidence of a relapse of the disease [2]. Factors predisposing individuals to the disease are also age (SM usually begins between the age of 20 and 40), and gender (in women, SM occurs two times more often) [9]. Multiple sclerosis seems to be a consequence of a set of the above factors coinciding.

The symptoms of multiple sclerosis. The symptoms of SM can be transient and sometimes difficult to describe. Typical symptoms characteristic of multiple sclerosis are the following: ocular ataxia, chanted speech, intention tremor – these are the so-called "Charcot's Triad". The most frequent symptoms of multiple sclerosis include: retrobulbar optic neuritis - after several weeks, nerve atrophy develops; symptoms of spinal cord damage which usually take the form of motor disorders – limb paresis (asthenia), as well as sensory disorders – numbness, formication, and limb paresthesia. In this case, Lhermitte's symptom is typical, i.e. electric tingling along the spine following neck flexion [3,10]. The pyramidal symptoms include increased tension, very strong reflexes and pathological symptoms, e.g. Babiński's reflex [3].

Other typical symptoms are disorders in urination – pollakisuria, quick urination, urinary incontinence or retention, or the loss of sexual drive and impotence [3,10]. The symptoms of brain stem damage present themselves with vision disorders, i.e. vision clouding and double vision, balance and coordination disorders, motor and sensory disorders of the face, *inter alia* peripheral seventh nerve palsy, dizziness, vomiting, as well as bulbar symptoms leading to dysarthria and swallowing disorders [3].

Cerebral symptoms occur in patients with a long pathological process and comprise a reduction of cognitive functions. Among the less frequent symptoms are hemiparesis and hemianopsia. Epileptic fits can also take place. Some patients also show behaviour disorders, emotional lability and depressive or manic states [3, 8, 10].

The fatigue syndrome. Well over half of the MS patients report having fatigue syndromes. These are characterised by uncontrolled apathy, dejection, indifference, exhaustion and the lack of energy. This is a very onerous symptom, and is the main reason for their inability to work and for their entering into social isolation. Immunological and neuro-endocrine factors can play a substantial role in the pathogenesis of fatigue. In the diagnosis of the syndrome, other causes must be excluded, such as the side-effects of medicines, infections, sleeping disorders, metabolic diseases [4].

Fatigue in the course of SM is increasingly frequently described in the literature on the subject. In two thirds of patients with SM, this is one of the main three symptoms. In the light of studies on SM, the lack of correspondence between fatigue and the degree of motor disability and depression is confirmed [6].

In the course of SM, several types of fatigue are distinguished:

- Fatigue Type 1 is described as typical physiological fatigue after effort, intensive work or active leisure activities, similar to that in the case of healthy people. However, these patients need more frequent and longer rest than healthy people.
- Fatigue Type 2 is a result of weakened physical stamina. Patients with SM, just as with healthy people without proper stamina, get tired easily.
- Fatigue type 3 is a neuromuscular fatigue or is connected with the so-called "short circuit mechanism". This means that neural fibres, damaged by demyelination, are stimulated when performing repeated activities, until exhaustion occurs. In such a type of fatigue, small pauses during the performance of certain activities make it possible to save energy and reduce the degree of fatigue.
- Fatigue type 4 is referred to as "psychogenic fatigue", as it is connected with depression. In order to reduce these symptoms, the application of anti-depression treatments is necessary.
- Fatigue type 5 manifests itself with feelings of overwhelming weariness even after slight effort in the case of patients without depression. It is also referred to as "fatiguability" or "SM-dependent fatigue syndrome". This

type of fatigue is diagnosed in 85–90% of patients and is commonly observed in SM. Most probably it is connected with demyelinatory lesions with the participation of immunological factors (cytokines: IL-1, IL-2, IL-6 [4].

REFERENCES

1. Adamczyk K.: Pielęgniarstwo neurologiczne. Wydawnictwo Czelej, Lublin 60, 2000.
2. Bartosik-Psujek H. and Stelmasiak Z.: Stwardnienie rozsiane - trudne odpowiedzi na proste pytania. *Neurologia i Neurochirurgia Polska* 5, 443, 2006.
3. Członkowska A.: Stwardnienie rozsiane – współczesna diagnostyka i leczenie. *Przewodnik Lekarza* 1, 7, 2003.
4. Dworzańska E., Mitosek-Szewczyk E., Stelmasiak Z.: Zespół zmęczenia w stwardnieniu rozsianym. *Neurologia i Neurochirurgia Polska* 1, 71–72, 2009.
5. Losy J.: Podstawy kliniczne chorób demielinizacyjnych – stwardnienie rozsiane. (w:) Jaracz K. and W. Kozubski, Pielęgniarstwo neurologiczne. Podręcznik dla studiów medycznych. Wydawnictwo Lekarskie PZWL, Warszawa, 250–252, 2008.
6. Opara J., Jaracz K. and Brola W.: Aktualne możliwości oceny jakości życia w stwardnieniu rozsianym. *Neurologia i Neurochirurgia Polska* 4, 339, 2006.
7. Palasik W.: Stwardnienie rozsiane – nowe tendencje terapeutyczne. *Przewodnik Lekarza* 9, 41–42, 2007.
8. Papuć E., Belniak E. and Stelmasiak Z.: Zaburzenia depresyjne u chorych na stwardnienie rozsiane. *Badania nad Schizofrenią* 5, 466, 2004.
9. Pierzchała K. and Kubicka K.: Rola czynników środowiskowych w patogenezie stwardnienia rozsianego. *Wiadomości Lekarskie* 1, 37–39, 2009.
10. Prusiński A.: Neurologia praktyczna. Wydawnictwo Lekarskie PZWL, Warszawa 311, 2001.