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# *Non-characteristic primary symptoms in the patient with sclerosis multiplex*

Multiple sclerosis (MS) is a chronic progressive demyelinating disease of the central nervous system (CNS) with some inflammatory features, affecting the brain as well as spinal cord. Early destruction of neuronal processes, i.e. axons is not obligatory, but when it occurs, reconstruction of damaged myelin is not reparable. This leads to formation of typical MS lesions, composed of astrocytes and further axonal loss.

The cause of origin of this unpredictable disease is still unknown and MS cannot be cured yet. Up to now, immunomodulatory therapy is the only one with documented efficacy in decreasing the frequency of relapses and slowing the progress of neurological impairment. As the inflammatory activity in MS is not confined to relapses or clinical impairment, and is often described before the first episode and usually goes even during remission, the immunomodulatory treatment is recommended, especially in the early stage of the process (1). We present a diagnosed, clinically proven MS patient (according to McDonald's criteria) (2), who was treated with Interferon at the early phase of the disease.

Patient J.J., age 25, has been completely healthy up to now. The first symptoms occurred in June 1998. The patient complained of right limb weakness, speech disturbance and walk disability. During hospitalisation in the Department of Neurology the performed neurological examination revealed mild motor dysphasia, slight right pyramidal hemiparesis, more affected in the upper limb and moderate decrease in touch and pain on the right side of the body. CT scans, extracranial carotid and vertebral arteries ultrasonography and visual-evoked potential test were normal. Vascular-active treatment (Fraxiparine, Dextran) as well as rehabilitation was introduced. At the time of discharge from our Department only slight right hemiparesis could be seen. The diagnosis was vascular lesion of the brain.

Next symptoms, such as walk disability and epileptiform seizures related to the right side of the body occurred in August 1998, just two months after the first hospitalisation. Repeated CT, standard EEG and sleep deprivation EEG test, as well as laboratory test (against toxoplasmosis, cysticercosis and echinococcosis) revealed no abnormalities. Standard cerebrospinal fluid (CSF) examination was normal, but oligoclonal bands were detected. Thus, MRI scans were performed. These revealed the presence of few periventricular lesions, and single focuses in the corpus callosum, left cerebellar hemisphere and the left side of the brain stem. Tegretol was added to current therapy. Diagnosis was reevaluated and was: *Foci organici multiplices cerebri et cerebelli ad observationem tardam. Paroxysmi epileptiformis ad observatione.*

In September 2001 the patient felt orbital pain in the right eye, especially during eye movement, and worse visual acuity. Optic neuritis was diagnosed. The applied treatment brought regression of symptoms.

Three months later, in December 2001 the patient was again hospitalised in our Department due to right limbs weakness and right side numb sensations. Neurological examination revealed right side hemiparesis with hyperreflexia and pyramidal signs. There was also a tendency to the presence of Babinski's sign on the left side. The diagnosis was MS and epilepsy in anamnesis (probably symptomatic). Steroid therapy were effective and clinical signs were reduced.

Next deterioration occurred in February 2002. For the first time cerebellar abnormalities were detected. Moreover, spastic paraparesis of lower extremities dominated in the clinical status. Steroid therapy was partially effective and neurological impairment gradually decreased. MRI scans (FSE, FAST and FLAIR) revealed periventricularly and within deformed and thinly corpus callosum bilateral numerous hyperintensities, affecting white matter and described as MS lesions. The major lesion had 2 cm diameter and was localised in the white matter of the right hemisphere.

In one of the described foci near the frontal horn of the right lateral ventricle there were seen disintegration features – apoplectic cavern with a signal similar to the cerebro-spinal fluid signal. EEG showed an abnormality like focal changes seen clearly in Hv activation, with groups of slow waves delta-theta, 3–5 Hz up to 70 μV, in the left anterior and the middle temporal lobe. Hyperventilation markedly activated the described abnormalities.

Two months later, in April 2002 the patient began interferon β-1b (Betaferon) therapy, with good tolerability. At the onset of therapy, during the first 3 months the patient complained of flu-like symptoms, i.e. fever, chills, muscular pain, a general feeling of being unwell, but the occurrence of these symptoms decreased over this time. After 2 years of interferon β-1b therapy good treatment tolerability and the patient's frame of mind were corroborated, no relapses were seen and neurological status was continuously stable, as well as no serious side-effect occurred.

MS often demonstrates wide heterogeneity in clinical history and clinical symptoms. On the basis of clinical status and other studies (MRI, CSF, visual-evoked potentials) we recognised relapsing-remitting course of MS. The patient started interferon β-1b therapy. Immunomodulatory therapy influenced the natural course of the disease. Interferon β-1b caused total reduction in relapses as well as a slower course of the disease and disability progress.

The onset of the disease can be mono-or multi-symptomatic, typical or non-typical. Speech disturbances belong to rare symptoms of multiple sclerosis. Usually they appear suddenly, often with incomplete or transient character and afterwards they generally cease. They may accompany right-sided hemiparesis in dextromanual subjects (3).

Generalised epileptic seizure can be the only clinical symptom of multiple sclerosis during relapse of the disease (4, 5). However, attacks may appear in every period of disease, independently of its natural course. Epileptic seizures occur parallel to other symptoms of the first bout of multiple sclerosis. Appearance of hemiparesis and epileptic seizures in patient may provoke to make a diagnosis other than MS. We encountered a similar situation in this case. Just after enclosing next symptoms met at successive bout of the disease and after extension of diagnostic explorations, the correct diagnosis became possible.

## REFERENCES

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## SUMMARY

Multiple sclerosis (MS) is a chronic progressive demyelinating disease of the central nervous system with some inflammatory features. The cause of origin of this unpredictable disease is still unknown and MS cannot be cured yet. Up to now, immunomodulatory therapy is the only one with documented efficacy in decreasing the frequency of relapses and slowing the progress of neurological impairment. We present a diagnosed, clinically proven MS patient (according to McDonald's criteria), who was treated with Interferon at the early phase of the disease. First, the patient complained of right limb weakness, speak disturbance and walk disability. Neurological examination revealed mild motor dysphasia and slight right pyramidal hemiparesis. CT scans were normal and the diagnosis was vascular lesion of the brain. Walk disability and eplieptiform seizures occurred two months after first hospitalisation. MRI scans revealed the presence of few periventricular lesions, and single focuses in the corpus callosum, left cerebellar hemisphere and the left side of the brain stem. Three years later optic neuritis was diagnosed and after the next three months the patient was again hospitalised due to right limbs weakness and right side numb sensations. Thus, the diagnosis was MS and epilepsy in anamnesis. Next deterioration occurred another three months later. For the first time cerebellar abnormalities were detected. MRI scans were typical of MS. The patient began interferon β-1b therapy. After 2 years' interferon β-1b therapy good treatment tolerability and the patient's frame of mind were corroborated, no relapses were seen and neurological status was continuously stable, as well as no serious side-effect occurred. Appearance of hemiparesis and epileptic seizures in the patient may provoke to make a diagnosis other than MS. Just after enclosing next symptoms met at successive bout of the disease and after extension of diagnostic explorations, the correct diagnosis became possible.

Niecharakterystyczne objawy początkowe u pacjenta ze stwardnieniem rozsianym

Opisano przypadek stwardnienia rozsianego, który rozpoczął się niedowładem połowiczym prawostronnym i zaburzeniami mowy. W następnym rzucie choroby wystąpił napad padaczkowy. Dopiero wykonanie badań pomocniczych, głównie MRI głowy, pozwoliło na postawienie rozpoznania. Częste rzuty choroby były wskazaniem do włączenie leczenia immunomodulującego interferonem beta 1-b. Uzyskano efekt leczenia pod postacią niewystępowania rzutów i ustabilizowania stanu neurologicznego pacjenta.