Trauma and multiple sclerosis
An hypothesis

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Summary. An obligatory event in the pathogenesis of the multiple sclerosis plaque appears to be an increase in the permeability of the blood-brain barrier. Neuropathological observations of the brain of persons suffering from concussion after relatively minor head injury, as well as of animals subjected to experimental brain injury, have shown that alterations of the blood-brain barrier constitute a common result of such trauma. It is postulated that the alterations of the blood-brain barrier secondary to trauma of the brain or spinal cord of patients with already established multiple sclerosis may result in an exacerbation or recurrence of a previously symptomatic plaque, in the appearance of symptoms from a silent lesion, or in the formation of a new plaque in such an area of selected vulnerability. In other persons injury to the nervous system may cause the development of multiple sclerosis plaques in the previously damaged areas when the disease has its onset after the trauma. There is no evidence to support the idea that trauma ever causes multiple sclerosis.

Key words: Trauma – Multiple sclerosis

Introduction

Although there is general agreement that physical trauma does not cause multiple sclerosis (MS), there is a lack of consensus regarding its effect on the exacerbations and the progression of the disease. McAlpine [23], Miller [26], Millar [25] and Walton [42] favor the idea that trauma must be considered a precipitating factor for exacerbations; Adams and Victor [1], Matthews [21], Kelly [16] and Hallpike [14] offer no definite opinion, while Keschner [17] is definitely against the idea. Bamford et al. [4], on the basis of their own prospective epidemiological study and a detailed review of previously published epidemiological studies of the problem, come to the conclusion that "...it is most unlikely that trauma is a primary cause of MS although it could be a triggering factor."

The exact pathogenesis of MS remains mysterious, but an alteration of the blood-brain barrier (BBB) is now known to accompany activity of the disease [31, 40]. This knowledge, coupled with better understanding of the effects of physical trauma on the nervous system, makes it advisable to examine the pathophysiological changes that may account for the observed worsening of symptoms in many MS patients who have suffered physical trauma.

Case reports

The following five brief case studies do not represent a cross-section of MS patients nor do they provide any kind of systematic survey. They are simply meant to illustrate a variety of scenarios of temporal relationship between trauma and the symptoms of MS which might suggest that the injury influenced the course of the illness.

Case 1

A 27-year-old woman suffered a mild flexion-extension injury of the neck when her car was struck from behind. Three days later she became aware of tingling and numbness of her left hand that soon involved the entire arm. Five years earlier she had had the same sensation in her left hand which had also lasted for about 5 weeks. Approximately 8 months after her minor automobile accident, she was in a second and more severe automobile accident, again causing a flexion-extension injury of the neck, but this time resulting in intense muscle spasm of the neck and acute pain in her neck and left arm. The patient again experienced numbness and tingling of her left hand 5 or 6 days later that rapidly affected the entire left side of the body. She noticed a difference in temperature sensation on the left side of the body, as well as a sensation of electricity going down the middle of her back whenever she flexed her neck quickly. She had had the same momentary electrical sensation two or three times in the previous few months. In the next weeks, she developed weakness and an inversion of her left foot as well as weakness of her right leg. Her gait became progressively more ataxic. Several months later she had an episode of right optic neuritis. Three months after the second accident, at which time her symptoms had essentially remained unchanged, an examination of the cerebrospinal fluid (CSF) revealed the presence of 25 lymphocytes, a total protein of 50 mg/100 ml and an immunoglobulin content (IgG) of 30%, but without oligoclonal bands.

Comment. This young woman appears to have had a high cervical spinal cord plaque that had been spontaneously symptomatic only once, but became symptomatic transiently after the first accident and permanently after the second, more severe accident 8 months later. Her MS progressed steadily from that time on, as evidenced by the appearance of signs and symptoms of lesions elsewhere in the nervous system.
Case 2
A 45-year-old man had had MS that started as a progressive paraparesis 14 years before he had an accident. Eleven years previously he developed a left optic neuritis that resolved spontaneously but that left him with a mild degree of optic atrophy and decreased visual acuity in the left eye. About 4 years later he had a 2-week period of diplopia and was diagnosed as having a right internuclear ophthalmoplegia. For the previous 3 years he had been confined to a wheelchair.

One day, while being transported in a chair car, his wheelchair was improperly secured. It tipped backward and he hit his neck and the back of his head on the floor of the van. He was unconscious for a few minutes. About 18 hours later he had severe weakness of both arms, which over the next few days became completely paralyzed. A neurological examination now showed new findings of flaccid weakness of the upper extremities with absent biceps, triceps and brachioradialis reflexes. His sensory level had moved up to C3 from the previous T8–9. Over the next several weeks he developed severe spasticity and hyperreflexia of his upper extremities. There was no indication in his previous medical history of any signs or symptoms referable to the upper cervical cord.

Comment. The rapidity with which the new signs and symptoms developed suggest that an asymptomatic plaque had been present in the cervical cord before the accident, although a direct injury to the cord rather than an activation of an MS plaque cannot be ruled out.

Case 3
A 35-year-old factory worker had been in apparently good health until he was hit behind the left ear by the heavy hook of an overhead crane at his place of work. He was unconscious for a few minutes. When he recovered, he complained of an intense headache and several hours later of severe dizziness. This was so incapacitating that he was unable to walk and his gait was distinctly ataxic. He had nystagmus in all directions of gaze and complained of severe diplopia, which was diagnosed as due to a bilateral internuclear ophthalmoplegia. The left side of his face was numb, especially in the chin area. In addition to the severe gait ataxia, he had dysmetria, incoordination, and dysdiadochokinesia of both arms and hands, more apparent on the left side. He also had mild left sixth and seventh nerve weakness. The patient continued to complain of severe dizziness and nausea and vomited several times. A CT scan of his head was normal as was the CSF examination, which showed no evidence of intracranial bleeding.

In the next few days the patient’s symptoms improved somewhat, but 2 months later he suddenly lost vision in the left eye. Shortly thereafter he had weakness and numbness of his right leg, as well as urinary frequency and urgency. Visual evoked responses showed a P-100 latency of 132 ms in the left eye; brainstem auditory evoked responses showed a markedly prolonged latency between waves IV and V on the left side and to a lesser degree on the right side. A CSF examination approximately 4 months after the accident showed 10 lymphocytes, a total protein of 56 mg/100 ml, IgG of 15.3%, and three oligoclonal bands.

Comment. Until the accident there was no clinical indication that the patient may have been suffering from MS, but the trauma seems to have brought to light one or more MS lesions in the brainstem. The subsequent course made obvious the fact that the patient had MS.

Case 4
A 32-year-old truck driver had had an episode of vertical diplopia lasting for 3 weeks that had been ascribed to fatigue and treated with an eye patch. About 7 months later, he got into a fight, was hit in the face, and fell backward, hitting the back of his head on the ground. He was dazed for a few seconds but got up immediately. He had no residua and returned to work. About 4 weeks later he began having difficulty controlling his left leg. Within a few days this difficulty involved his right leg as well. He also felt tingling and numbness across his lower abdomen for about a week which later disappeared. At about this time he noted some problems with handwriting, as well as clumsiness of both hands, slowing of his speech and increasing impairment of balance. A CT scan was obtained that proved normal. A neurological examination showed him to have severe ataxia of gait with marked dysmetria, incoordination, and bilateral dysdiadochokinesia. His position and vibratory senses were normal, and he had bilateral horizontal nystagmus. His reflexes were generally equal but slightly diminished and he had no pathological reflexes.

When seen again several months later, the patient had developed weakness and spasticity of the lower extremities as well as urinary frequency and urgency with occasional incontinence. In addition to his earlier neurological abnormalities, he had developed hyperreflexia in the lower extremities along with bilateral Babinski signs, decreased position and vibratory senses in his legs, and a poorly defined sensory level to pinprick at about T10.

Comment. In retrospect, the patient probably had had a previous episode of MS manifested by diplopia. Because of the time lag between the accident and development of the cerebellar symptoms, it is postulated that a new plaque had formed in the area of the injury to the cerebellum resulting from the blow to the face and the fall to the ground. The mechanism by which a cerebellar lesion may result from a blow to the face, and other types of contrecoup injuries, are well illustrated in the study by Lampert and Hardman [19] on brain injuries in boxers.

Case 5
A 45-year-old housewife was in an automobile accident in which she suffered a flexion-extension injury. She received treatment for neck and low back pain and muscle spasms resulting from the accident. Over the next 2 months she reported stiffness and weakness of the left leg. She also complained of left arm pain radiating into her hand. Electromyographic examination showed left C5 and S1 radiculopathies. Myelography revealed a large spondylotic ridge at the C4–5 interspace as well as some lumbar osteoarthritides. About 26 months after her accident the patient underwent anterior cervical disectomy and fusion at the C4–5 interspace. Just before her operation she had generalized hyperreflexia with mild but definite spasticity in all four extremities and bilateral Babinski signs. Some fasciculations were noted in the shoulder girdle musculature. Shortly after surgery her gait improved and her right plantar response became flexor although the left one remained extensor. The fasciculations were no longer visible, but the reflexes remained hypoactive in her lower extremities.