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DEMYELINATING DISEASE OF THE CENTRAL NERVOUS SYSTEM – MULTIPLE SCLEROSIS.

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Abstract: This article discusses the causes, pathogenesis, and clinical features of multiple sclerosis. The process of demyelination, which underlies the disease and involves damage to the myelin sheath surrounding nerve fibers, is explained from a scientific perspective. In addition, the roles of genetic factors, viral infections, vitamin D deficiency, and harmful habits in the development of the disease are analyzed. The clinical types of multiple sclerosis, their course, and main symptoms are systematically described. The article also provides information about diagnostic methods, including modern diagnostic approaches, as well as principles of treatment. The main aim of this work is to summarize knowledge about multiple sclerosis, explain its complex nature in a clear and understandable way, and highlight the importance of early diagnosis and effective disease management.

Multiple sclerosis is a demyelinating disease of the central nervous system, characterized by microscopic structural changes in the tissues of the brain and spinal cord. To fully understand this condition, it is important to study it not only from a clinical perspective but also from a histological point of view. All processes in the human body — from moving a fingertip to solving complex mathematical problems — are controlled by the nervous system. This system is the fastest and most sophisticated communication network in the body, and every movement, sensation, and thought depends on its continuous function. The basic structural and functional unit of the nervous system is the neuron. Each neuron acts like a small “station” that receives, processes, and transmits information. Its central part, the perikaryon (cell body), is responsible for essential cellular functions. Dendrites receive incoming nerve impulses, while the axon transmits these impulses to other cells. A particularly important structure is the myelin sheath surrounding the axon, which ensures the fast and accurate conduction of nerve impulses. Myelin not only provides protection but also significantly increases the speed of signal transmission. The cells responsible for forming this sheath, especially oligodendrocytes, are essential for the normal functioning of axons. When myelin is intact, the nervous system functions smoothly and efficiently. However, under the influence of various factors, a process called demyelination begins. In this process, the myelin sheath is damaged or destroyed, leading to impaired transmission of nerve impulses. These microscopic changes can be identified histologically and play a key role in understanding the underlying nature of the disease.

Multiple sclerosis was first scientifically described in the 19th century by the French scientist Jean-Martin Charcot. He identified that, as a result of myelin destruction, scar-like changes develop in nervous tissue. This was an important discovery that helped explain the histological basis of the disease. Thus, understanding the normal microscopic structure of the nervous system serves as a foundation for recognizing the pathological changes that occur in multiple sclerosis. Studying these histological changes allows for a deeper understanding of the

disease's development mechanisms, as well as contributes to its early diagnosis and more effective management.

The brain is surrounded by small blood vessels called capillaries. Through these capillaries, the brain receives the nutrients it needs. These blood vessels consist of an endothelial layer and a basal membrane. Between these layers there are tight junctions, which help regulate what can pass through the vessel wall. In addition, astrocyte cells surround the blood vessels with their processes from the outside. Together, these structures form the blood-brain barrier (BBB). The blood-brain barrier is not just a simple barrier; it is a complex "filter" and "guardian" of the central nervous system (CNS). It plays a crucial role in maintaining the internal stability of the brain, known as homeostasis. One of the main functions of the BBB is protection. It prevents harmful substances such as microbes, toxins, and dangerous chemicals in the blood from entering brain tissue. At the same time, it has selective permeability, allowing essential substances for the brain — such as oxygen, glucose, and amino acids — to pass through, while blocking unnecessary or harmful compounds. In addition, the BBB also performs an immune regulatory function. Under normal conditions, it strictly limits the entry of large immune cells such as T-lymphocytes and antibodies, because the brain is considered an immune-privileged site. However, in certain conditions, the integrity of the BBB may be compromised. This can occur due to inflammatory processes, oxidative stress, or neuroviral and bacterial toxins. As a result, the "filtering" function of the BBB is disrupted, and cells that normally cannot pass through begin to enter brain tissue. Among these are T-lymphocytes. Due to certain abnormal immune responses, T-lymphocytes mistakenly recognize the myelin sheath as a foreign substance. After this, immune cells begin to attack the myelin sheath. As a result, the body's own immune system destroys its own myelin. For this reason, the disease is also classified as an autoimmune disorder. Immune cells at the affected site recruit other immune cells, such as B-lymphocytes, leading to inflammation in that area. During inflammation, various biologically active substances — cytokines and antibodies — are released. Under their influence, the myelin sheath surrounding nerve fibers begins to deteriorate. This process is called demyelination. Initially, the axon itself may remain intact, but the transmission of nerve impulses becomes significantly slowed or disrupted. At this stage, early clinical symptoms such as blurred vision and muscle weakness may appear.

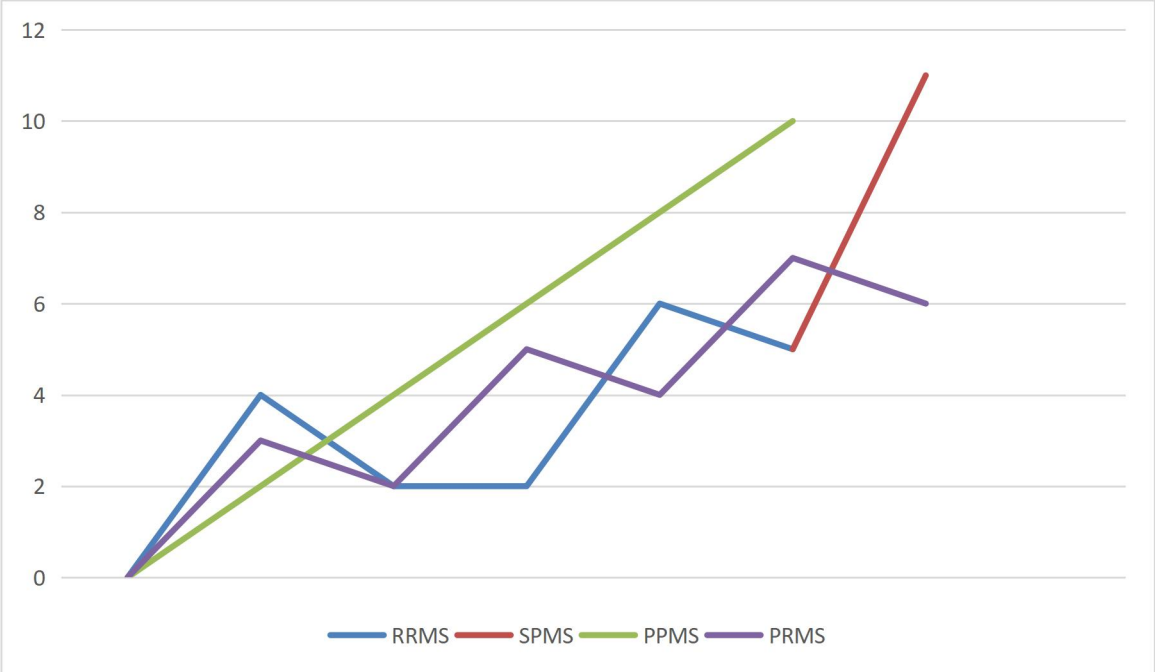
In multiple sclerosis, the disease does not always follow the same pattern; it alternates between periods of worsening and remission. The stable or recovery phase is called remission. During remission, a certain degree of recovery occurs in the body. In this process, oligodendrocytes play an important role, as they attempt to regenerate the myelin sheath around nerve fibers. If this process is successful, nerve signal conduction improves and the patient may feel better. However, this repair is not always complete. Sometimes the newly formed myelin is not sufficiently strong, or it may not fully regenerate at all. A relapse or exacerbation of the disease is associated with renewed activation of the immune system, which again attacks the myelin in nerve fibers. This leads to a new demyelination process, and previous symptoms may return or new ones may appear. Therefore, multiple sclerosis progresses with alternating periods of improvement and worsening. Once a "gap" is formed in the area of myelin loss, the body attempts to repair it. In this process, astrocytes become activated. They accumulate in the damaged area and try to limit inflammation and "seal" the tissue. This stage is called reactive gliosis. However, this repair process is not always beneficial. When astrocytes proliferate excessively, a dense fibrous scar-like tissue forms in the affected region. This condition is known as sclerosis, or scarring. These sclerotic lesions can appear in different parts of the brain, which is why the disease is called multiple (disseminated) sclerosis. In severely affected areas, the transmission of nerve impulses may be significantly or even completely blocked. Thus, the

progression of multiple sclerosis can be summarized as follows: immune attack → inflammation → myelin destruction → gliosis → sclerosis. For this reason, modern treatment approaches are primarily focused on the early stage of the disease—controlling and suppressing inflammation. If this stage is properly managed in time, the development of severe and irreversible complications can be prevented.

Multiple factors play an important role in the development of multiple sclerosis, including genetic predisposition, vitamin D deficiency, viral infections, and harmful habits. Among these, genetic factors create a susceptibility to the disease, but multiple sclerosis is not directly inherited. In other words, genes increase the risk, but they do not guarantee that the disease will occur. Studies on twins show that the risk of developing the disease is about 25–50% in identical twins, while in fraternal twins it is around 5–10%. This indicates that although genetic factors are important, environmental influences also play a significant role. One of the most important genetic factors is the HLA-DRB1 gene located on chromosome 6. This gene regulates the immune system, and its variations may cause the immune system to mistakenly attack the myelin sheath. Infectious factors are also important in the development of multiple sclerosis. Some viruses can "mislead" the immune system, causing it to react against the body's own tissues. This happens due to molecular mimicry, where viral proteins resemble the body's own proteins, leading the immune system to attack healthy cells as well. The most studied virus in this context is the Epstein-Barr virus, which has been found in nearly all patients with multiple sclerosis. Other viruses, such as measles, herpes, and mumps, are also considered possible contributing factors. These viruses do not directly cause the disease but may trigger its onset. Another key factor is vitamin D deficiency. Multiple sclerosis is more common in regions with limited sunlight exposure. Vitamin D helps regulate the immune system and controls the activity of T-lymphocytes. A deficiency can lead to excessive activation of these cells, causing them to attack the myelin sheath. Harmful habits, especially smoking, also contribute to the development of the disease. Smoking disrupts the immune system, increases inflammation, and accelerates damage to the myelin sheath. As a result, the disease may progress more rapidly, become more severe, and increase the risk of disability. Therefore, quitting smoking is an important preventive measure.

Multiple sclerosis is a clinically variable disease, and according to modern classification, it has four main types: relapsing-remitting (RRMS), secondary progressive (SPMS), primary progressive (PPMS), and progressive-relapsing (PRMS). These types reflect the pattern of disease onset and progression. The most common form is relapsing-remitting multiple sclerosis (RRMS), which occurs in about 80–85% of patients at onset. This type is characterized by alternating periods of relapse (exacerbation) and remission. During relapses, the immune system attacks the myelin sheath, causing demyelination and leading to symptoms such as visual impairment and muscle weakness. During remission, inflammation decreases and symptoms improve, although recovery is often incomplete. This form usually begins around the age of 20 and is more common in women. Over time, RRMS often progresses to secondary progressive multiple sclerosis (SPMS). This typically develops within 15–20 years and is observed in about 60–65% of untreated patients. At this stage, the disease gradually and continuously worsens, while remissions become less frequent or disappear. In addition to demyelination, the nerve fibers themselves are damaged, leading to an accumulation of neurological deficits and increasing disability. Primary progressive multiple sclerosis (PPMS) is less common, accounting for about 10% of cases. In this form, the disease progresses continuously from the beginning, with little or no remission. The process is characterized more by slow nerve fiber degeneration than by inflammation, and motor function gradually declines. It usually begins after the age of 30 and affects men and women equally. Progressive-relapsing multiple sclerosis (PRMS) is the

rarest type, occurring in less than 5% of patients. In this form, the disease progresses steadily but is also accompanied by periodic relapses. After each relapse, the patient's condition gradually worsens. It typically begins between the ages of 30 and 50. Below is a schematic representation of the clinical course of multiple sclerosis. Upward lines indicate disease exacerbation (demyelination), while downward lines represent a reduction in symptoms, i.e., remission.



Clinical types of multiple sclerosis

Type	Frequency	Age of onset	Gender ratio	Course
Relapsing Remitting (RRMS)	80-85%	Around 20	More common in women than men	Periods of exacerbation and remission alternate.
Secondary Progressive (SPMS)	Develops in 60-65% of RRMS cases	10-15 years after RRMS onset	Becomes more equal between genders	Remissions shorten and gradually worsen over time
Primary Prograssive (PPMS)	10%	Around 30+	Equal in men and women	Continuous progression from the beginning without clear remissions
Progressive Relapsin (PRMS)	<5%	30-50	Slightly more common in women	Continuous worsening with occasional relapses

Multiple sclerosis is a disease that presents with a wide range of clinical symptoms, which vary depending on the part of the central nervous system affected. In the early stages, symptoms

are often temporary and may later recur. Visual disturbances are among the earliest and most common signs. Patients may experience blurred vision, reduced vision in one eye, or pain during eye movement. In some cases, double vision (diplopia) may also occur. Motor symptoms are also common and include muscle weakness, fatigue, stiffness, and spasms. Over time, patients may develop difficulties with walking and loss of balance. These changes are related to impaired transmission of nerve impulses and are a result of the demyelination process. Sensory disturbances play an important role as well. Patients often report numbness, tingling, prickling sensations, or burning pain in the hands and feet. In some cases, there may be reduced sensitivity in certain parts of the body. Additionally, problems with balance and coordination may occur, such as dizziness, unsteady gait, and impaired coordination of movements. General symptoms include severe fatigue, reduced concentration, and sometimes slowed speech. Some patients may also experience bladder dysfunction. An important feature of the disease is that symptoms may decrease or even disappear for a period of time. This is associated with the remission phase, although symptoms may return later. In summary, the symptoms of multiple sclerosis can affect multiple systems and tend to change depending on the stage and progression of the disease.

Multiple sclerosis cannot be diagnosed with a single test or examination. Instead, it requires a comprehensive approach that combines several methods. In the diagnostic process, not only clinical symptoms but also the underlying histological changes of the disease are important. Patient complaints and neurological examination findings are essential, especially when symptoms show a relapsing pattern – that is, periods of remission and relapse. This pattern suggests the demyelinating nature of the disease. One of the main diagnostic tools is MRI (magnetic resonance imaging), which provides a macroscopic view of demyelinating lesions that occur at the histological level. In other words, areas where myelin has been damaged at a microscopic level can be visualized on MRI scans. In addition, analysis of cerebrospinal fluid can reveal oligoclonal bands, which reflect immune system activity and indirectly confirm the presence of inflammation at the histological level. Another important method is evoked potentials testing, which evaluates the speed of nerve impulse conduction. This helps detect functional impairments at the level of axons and myelin. In summary, diagnostic methods for multiple sclerosis are designed to identify structural and functional changes in nervous tissue, either directly or indirectly.

Treatment of multiple sclerosis is essentially aimed at influencing these underlying histological processes. The main goals of therapy are to reduce inflammation and slow down the destruction of myelin. During relapse episodes, corticosteroid medications are used to decrease inflammation in nervous tissue and limit damage at the cellular level. Disease-modifying therapies help regulate the immune system and prevent the formation of new demyelinating lesions, thereby slowing disease progression. Symptomatic treatment and rehabilitation are also important, as they help preserve the functional state of the nervous system. Physiotherapy and regular exercise can support partial compensation of damaged neural pathways. Although these approaches do not fully restore histological structures, they help maximize the functional capacity of the remaining nerve fibers and improve the patient's quality of life.

In conclusion, multiple sclerosis is a complex, multifactorial disease of the central nervous system that primarily develops at the histological level. Its core mechanism is a dysfunction of the immune system, which leads to damage of the myelin sheath—known as the process of demyelination. Over time, these changes may progress to gliosis and sclerosis. The clinical forms and symptoms of the disease arise as a direct result of these microscopic changes. Diagnostic methods are aimed at identifying these underlying histological alterations, while treatment strategies focus on slowing and controlling them. Therefore, a histological perspective

is essential for a deeper understanding of multiple sclerosis, as it allows for earlier diagnosis, more accurate assessment, and more effective management of the disease.

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