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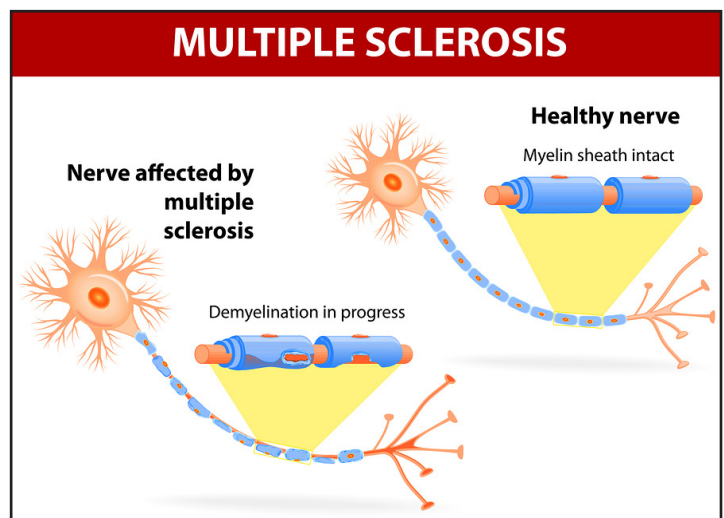
“Multiple Sclerosis--Pharmacy Perspective”

February 2015

Is incidence of MS increasing? It seems like it. Maybe it's just that there is more information available. Our goals in this lesson are to update this topic, and discuss current treatment trends.

Pharmacists will be able to:

1. Define MS & state its pathogenesis.
2. List types of MS.
3. Describe symptoms of MS.
4. State methods used to diagnose MS.
5. List medications used in treating MS.
6. Discuss side effects associated with MS treatments.



Technicians will be able to:

1. Define MS.
2. Classify MS.
3. State methods used in MS diagnosis.
4. Describe the prognosis for MS patients.

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MULTIPLE SCLEROSIS (MS)

Multiple Sclerosis (Sclerosis is derived from the word sclera meaning lesions or plaque) is an inflammatory disease of the central nervous system (CNS) that results in degeneration of the myelin and formation of plaques in the brain. This leads to the disruption of communication from the brain to the rest of the body. Myelin is the protective sheath consisting mainly of fat and protein. It covers and insulates the cells of the brain and the spinal cord. The inflammatory process is believed to be autoimmune, which will eventually cause an irreversible and gradual damage to the nerve cells. This then gives rise to the emergence of various and unpredictable neurological signs and symptoms. The peripheral nervous system is usually unaffected.

EPIDEMIOLOGY

MS is considered the most common neurological disease that impacts young adults. It affects individuals of all ages and races, but the most vulnerable are those between the ages of 20 to 50. It is more common among women than men at a ratio of 2 to 1. There are over 2.5 million cases worldwide, 400,000 of which are in the U.S. This disease can strike children. About 2% - 5% of patients start experiencing symptoms prior to reaching age 18. Most data points to environmental and genetic factors that affect the immune system. Environmental factors usually occur during childhood. The first symptoms arise shortly after exposure to such an environmental factor. The exposure period is followed by an absence of symptoms that lasts until the patient reaches adulthood. It has been estimated that there are 8,000 to 10,000 children in the U.S. who suffer from MS, and 10,000 to 15,000 children who have had at least one symptom that may imply the presence of MS. Data suggest that MS is more common in areas of the world that are farthest from the equator. Thus the incidence appears to be higher in cold climates than warm ones. In the U.S., incidence of MS in the southern states is estimated to be about 57 – 78 cases per 100,000 individuals. In the northern states the rate is approximately from 110 – 140 cases per 100,000. Native Americans and Asian Americans have the lowest rate, whereas Caucasian Americans of Northern European decent have a higher risk of acquiring MS than indigenous populations. This indicates a relationship between ethnicity and geography. Moreover, moving from one area of the world to another appears to increase or decrease the risk of MS. Lastly, family members of MS patients develop the disease at about 10 – 50 times more than the general population.

PATHOGENESIS

The main pathological features of MS include:

1. Presence of plaques in the brain surrounded by white matter of the optic nerve, brain stem, basal ganglia and spinal cord.
2. Inflammation which may be a result of autoimmunity.
3. Damage to the myelin.

The plaques have a gray or pink color that can be differentiated from the white matter. The peripheral nervous system is usually unaffected. The disease occurs as a result of loss of oligodendrocytes, which are cells that build the myelin. Once the inflammatory process begins, the number of oligodendrocytes gradually decreases resulting in thinning of the myelin

and eventual loss of its structure. Following this damage, the neurons fail to conduct the electrical charges between the brain cells or between the brain and other parts of the body. Plaque forms around the destroyed axon. The plaques range in size from 1 or 2 mm to several centimeters. Once the plaques develop, macrophages or microglial cells (macrophages found in the brain and spinal cord) will act to remove myelin debris. This is followed by an increase in the number of glial cells causing gliosis, a change of the glial cells in response to damage to the CNS. Glial cells are non-neuronal cells that protect and support the neurons in the brain. The lesions themselves are composed of completely demyelinated cells, gliosis, and absence of oligodendrocytes.

The inflammatory process is triggered by T-cells that are T-lymphocytes, a type of white blood cell that is involved in cell-mediated immunity. They protect the body from infectious diseases and other invading entities. Once the blood-brain barrier is disrupted, the T-cells are able to enter the brain. The T-cells consider the myelin to be a foreign substance and attack it, thereby triggering auto-reactivity (autoimmunity). The inflammatory process then can interfere with the communication between the CNS and other parts of the body. Following invasion of the myelin, an inflammatory process triggers the release of factors such as cytokines (chemokines, interferon, interleukins, lymphokines) that are produced by various immune cells such as macrophages, lymphocytes, T-lymphocytes and mast cells. Additionally, antibodies like immunoglobulin (IgE) can be generated. The cytokines are important in the immune system. They are believed to play a role in the response of a host to infections, immune response, inflammation and cancer.

CAUSES

The precise causes are unknown. However, it has been speculated that environmental, childhood infections, immunologic and genetic factors may contribute to MS.

Geographical and environmental factors such as living in areas that are far from the equator seem to play a role in the occurrence of MS. On the other hand, people who live closer to the equator are exposed to plenty of sunshine resulting in the formation of vitamin D, a substance believed to play a role in protecting the body against immune-mediated diseases such as MS.

It has been reported that smoking not only increases the risk of the development of MS, but may increase intensity and hasten progression of the disease.

Viral and bacterial infections have been suggested as possible triggers because they can cause inflammation and myelin damage. There is some association between acute disseminated encephalomyelitis (ADEM) and acute hemorrhagic leukoencephalitis (AHLE) and MS. The aforementioned diseases can cause symptoms similar to those of MS, such as the formation of plaques.

The damage caused to myelin by MS is due to immune-mediated responses. However, what triggers this response and its exact mechanism is not clear.

While MS is not a hereditary disease, the risk of acquiring MS appears to be higher in close relatives such as a parent or a sibling.

Other factors that may increase the risk of developing the disease include: age, sex, race, and presence of autoimmune diseases.

DISEASE COURSE (TYPES)

There are four types or patterns of MS according to progression of the disease over time. Identification of the type is relied upon for diagnosis or treatment. The four types are:

1. Relapsing – remitting MS (RRMS)
2. Secondary – progressive MS (SPMS)
3. Primary – progressive MS (PPMS)
4. Progressive – relapsing MS (PRMS)

Relapsing-Remitting MS, which usually represents the onset of the disease, is the most common type, and affects about 85% of the patients who are initially diagnosed. The early symptom is characterized by neurological attack (relapse) as a result of myelin damage. The relapse develops over days or weeks, and is often followed by partial or complete absence of symptoms (remitting) that can last for months or years with no apparent worsening.

Secondary-Progressive MS course often follows RRMS and occurs as a result of progression of the symptoms. It occurs in 60% of patients who had RRMS. Most patients with RRMS will transition to SPMS. Deterioration of nerve cells and emergence of symptoms may occur without any definite periods of remission. However, remission does occur occasionally.

Primary-Progression MS presents itself by increased intensity of disability. The rate of progression varies from occasional, to minor or no remissions and improvement.

Progressive-Relapsing MS is a form of the disease where there is steady progression from the onset, leading to worsening of the neurological symptoms. The condition continues to deteriorate without remission.

SIGNS AND SYMPTOMS

Since MS affects the CNS and causes disruption of signals and communication. Patients may experience a wide range of neurological signs and symptoms that vary in intensity depending on the extent, and location of damage to the myelin. The symptoms vary from patient to patient. Some patients have permanent symptoms, while others may experience long periods of remission with no symptoms. A patient may complain of difficulty walking while another is able to walk normally. Locations of the plaques in the brain play an important role in the type of neurological signs and symptoms. The most commonly encountered symptoms usually cause autonomic, visual, motor, and sensory problems. Autonomic problems usually involve the heart, digestive and respiratory rates, urinary retention, urinary incontinence, erectile dysfunction, constipation, salivation, and difficulty in swallowing. Visual difficulties involve the occurrence of nystagmus (limited vision caused by involuntary eye movement), inflammation of the optic nerve and partial or complete vision loss. Motor problems exhibit as muscle weakness, muscle spasm, fatigue, tremor, dizziness, strong reflexes, hyper excitability of muscles, ataxia (difficulty in coordination of movement), problems with muscles that assist in speech or pronunciation of words (dysarthria), slurred speech and cognitive problems such as memory loss, and depression. Sensory problems are associated with tingling, numbness, a pricking sensation and itching.

The early symptoms usually consist of blurred or double vision, cognitive problems, balance loss, numbness and tingling in the extremities, trunk and face, and weakness in the legs. Fatigue which may interfere with daily life occurs in 80% of patients. Numbness or tingling in the trunk, extremities or face is considered the first warning signs. Muscle weakness is caused by damage to the nerves that control the muscular system. Dizziness is characterized by light-headedness, balance loss, and feeling faint. Sexual arousal may be impaired due to damage in the CNS along with the fatigue, spasticity and psychological or emotional difficulties. Pain is experienced by many MS patients. It has been estimated that 55% of MS sufferers experience pain and that 25% have chronic pain. The stressful life that accompanies MS may cause emotional changes such as increased irritability, mood swing, depression and occasional crying or laughing episodes. The presence of ataxia along with muscle weakness, fatigue, and hyper excitability of muscles may lead to walking problems. Spasticity or muscular stiffness from muscular contraction can lead to additional walking difficulties. Vision problems, which are the early signs of MS, are due to damage to the optic nerve. Urinary retention and incontinence occur in about 80% of patients. Constipation is another bothersome symptom. Speech difficulties become more prominent in the last stages of the disease and during periods of stress and fatigue. Tremors in various parts of the body occur depending on the damaged nerves that control movement, and location of the plaques. Damage to the nerves that control pectoral muscles can result in difficulty in breathing. Likewise, damage to nerves that control muscles in the mouth and throat may result in dysphagia (difficulty in swallowing). Seizures occur in about 2.5% of patients due to damaged parts of the brain resulting in disruption of electrical impulses generated within the brain. About 6% of MS patients report hearing impairment. Itching, burning, tingling or pricking sensations may also occur.

DIAGNOSIS

There are no definitive methods to diagnose MS. However, early determination is important as it may lead to slowing the progression of the disease. Symptoms, especially the early ones, mimic many other neurological disorders. The first helpful step is to rule out presence of such disorders. Patient medical and family histories should be studied. MS can be confirmed by:

1. presence of damage to two areas in the CNS, namely the brain, spinal cord, and the optic nerve,
2. evidence that the damage has occurred one month apart;
3. ruling out the presence of mimicking neurological disorders,
4. medical imaging and laboratory tests. Blood tests can help in the elimination of the existence of infection and inflammatory diseases. Lumbar puncture (spinal tap) can reveal the presence of abnormalities in white blood cells and antibodies that accompany MS.

The use of Magnetic Resonance Imaging (MRI) is a non-invasive method that provides a sensitive test for exploring CNS damage and confirmation of the presence of plaques or scars on the brain and spinal cord. Physical examination that shows the magnitude of nerve reflexes, the ability to move parts of the body, the presence of sensation, and vision strength are considered important in the diagnosis of MS. Eye examination includes pupil responses, changes in visual field (extent at which objects can be seen peripherally), blind spots in vision halos, vision loss or impairment, and rapid eye movement. Musculoskeletal

symptoms such as numbness, muscular spasm, difficulty in moving the extremities, problems in walking, coordination of movement, tremor, weakness in one or more arms and legs should be investigated. Finally, some tests are used to determine the electrical activity in certain areas of the brain in response to stimulation. Results indicate the presence or absence of irregularities or damage to certain nerves.

TREATMENT

Currently there are no cures. The main objective of treatment is to stop or reduce its progression, modify its course, alleviate symptoms, enhance recovery of relapses and improve quality of life, emotional health and wellness. Most cases involve the counsel of a neurologist and the participation of other specialists. In some mild cases no pharmacological treatment is necessary. However, continuous care and observations are important in order to assess possible progress of the disease. Once MS is diagnosed, care of the patient should last a lifetime. There are a number of drugs that have been approved by the FDA and are intended to check or slow progression, in particular the relapsing.

Interferon beta-1a is a cytokine produced by mammalian cells which act by reducing the inflammatory cells that enter the brain. This leads to reduction of nerve cell inflammation. Furthermore, it is believed that this drug improves the formation of nerve growth factor which is a protein necessary for growth and maintenance of nerve cells and continuation of their existence. It has been estimated that it reduces relapses by 30%, but about 30% of patients do not respond to treatment with interferons beta 1-a. The drug is administered parenterally. Adverse effects include: irritation and bruising at the subcutaneous injection site, flu-like symptoms, fever, fatigue, muscle aches, and headache. Many of these symptoms are transient. Monitoring the patient during therapy is necessary. Reports of slowing progression of disability are debatable.

Interferon beta-1b is a cytokine produced in modified E.coli. It has both a mechanism of action as well as adverse effects similar to those of interferon beta-1a.

Glatiramer acetate is believed to act by blocking the body immune system from attacking its own myelin (immunomodulator). The FDA has approved its use for the purpose of reducing the frequency of attacks, but not for slowing progression of disability. It is injected subcutaneously. Adverse effects include: inflammation at the injection site, dyspnea, flushing and rash shortly after injection. It is approved for treating SPMS.

Mitoxantrone is an antineoplastic drug used in the treatment of various types of cancer and in slowing progression of SPMS as well as prolonging the intervals between relapses in RRMS and PRMS. Its side effects include: nausea, vomiting, hair loss, immunosuppression, and damage to the heart. It is used only in severe advanced MS. It has been reported that mitoxantrone moderately slows progression of the disease and reduces appearance of attacks over two years. Because of its adverse effects, the drug is used only in patients whose ailment is severe and does not respond to other treatments.

Natalizumab is a humanized monoclonal antibody that is administered by intravenous infusion every four weeks. It acts by preventing the passage of inflammatory immune cells from the bloodstream, through the blood-brain-barrier and to the brain and spinal cord. It is effective in reducing the symptoms of MS, prevents relapses and vision impairment, and

slows cognitive deterioration. The main side effect is its vulnerability to progressive multifocal leukoencephalopathy (PML), an opportunistic viral infection of the brain. This condition occurs if natalizumab is given concurrently with interferon beta-1a. In Europe the drug is approved only as monotherapy. The drug is given to patients who have severe active MS, or do not respond to other medications.

Alemtuzumab is a recombinant, DNA-derived, humanized monoclonal antibody. In addition to its use in MS, it is employed in the treatment of chronic lymphocytic leukemia, and organ transplantation. It has been reported that the drug is helpful in treating MS and may reverse the detrimental effects of the disease. At the present time studies are being conducted to find out if synergistic effects can be achieved when alemtuzumab is used in combination with glatiramer acetate. The drug is contraindicated in the presence of systemic infections, and immunodeficiency diseases such as HIV. Adverse effects include: development of opportunistic viral infections, hypotension, fever, shortness of breath, bronchospasm, cardiac arrhythmia, myocardial infarction, and cardiac arrest.

Dimethyl fumarate is a derivative of fumaric acid found in bolete mushrooms and fumaria, a plant in the family papaveraceae. It has been reported that the drug is capable of reducing relapse rates and slows progression of the disease. The exact mechanism of action is unknown, but it is believed that the drug and its metabolites activate a nuclear factor pathway. In addition it acts as a nicotinic acid receptor agonist. It is administered orally twice daily. Adverse effects include: nausea, vomiting, diarrhea, flushing and decreased white blood cell count.

Teriflunomide is an active metabolite of leflunomide which acts as an immunomodulating drug that inhibits pyrimidine de novo synthesis. It inhibits rapidly dividing cells including T-cells which play a role in MS occurrence. Adverse effects include: liver damage, hair loss, nausea, vomiting, and diarrhea. Birth defects may occur if the drug is taken during pregnancy. Teriflunomide is taken orally once daily with or without food.

Fingolimod is an immunomodulating drug which has been reported to reduce the rate of occurrence of relapses as well as their intensity. It is a metabolite of the fungus *Isaria sinclairi*. It is an orally administered drug intended for reducing relapses and slowing the occurrence of disability of patients who suffer from relapsing-remitting MS. Main side effects are: vulnerability of the patient for infections, bradycardia, flu-like symptoms, headache and fatigue.

Corticosteroids are used for the control of acute attacks. They can be administered either intravenously (methyl prednisolone) or orally (prednisone). These medications are intended to reduce nerve inflammation but do not have influence or change the course of the disease.

Plasmaphoresis or plasma exchange may be attempted in acute attacks that do not respond to corticosteroids. This process involves removal of blood plasma and separation of blood cells. The blood cells are mixed with albumin solutions and then placed back into the patient's body.

SYMPTOMATIC TREATMENT

Treatment of symptoms and signs that occur during the course of MS need to be dealt with to improve quality of life. Physical therapy can help to improve impairments, disabilities, mobility

and functional ability. The physical therapist may interact to teach exercises at home either alone or with assistance from a family member. Furthermore, a physical therapist may teach the patient or family members how to use devices that are beneficial for performance of daily life activities. The therapy should begin immediately after diagnosis. Stretching tends to reduce muscle spasm and maintain muscle strength. This can assist to decrease balance problems, improve coordination, and diminish fatigue and pain. The therapist should train the patient on how to use canes, crutches, and wheelchairs, if needed. Problems affecting the GI and urinary tract may be encountered due to disruption of signals between the brain and the muscles that control urination and emptying the bowel. These need to be dealt with by using medications or taking preventative measures that prevent or reduce such problems. Analgesics are helpful in reducing pain. Muscle relaxants can reduce spasms and stiffness. Clinical studies have shown that the use of medical marijuana may slow the degeneration of the CNS of some patients with MS.

PROGNOSIS & SUMMARY

MS has no cure. Early diagnosis, use of medications, life-style changes, proper diet and physical therapy can slow progression of the disease and reduce the number and severity of relapses. Progression of MS varies from one patient to another. Some individuals may feel healthy even though the disease is progressing. Others may have frequent relapses, and others rarely encounter attacks. Progress of the disease depends on factors such as gender, age, early diagnosis and start of therapy, the severity of disability and the type of MS. It has been estimated that the average life expectancy of an MS patient from the initial diagnosis is 30 years. About 65% of deaths occur as a result of complications and or pulmonary challenges as a result of weakness of the ventilator muscles, infections such as pressure sores, urinary tract infections, aspiration pneumonia resulting from swallowing problems and suicide which is found to be 7.5 times more than for the general population.

MS is an insidious, unpredictable neurological disease that is believed to be due to autoimmunity that results in damage to the myelin. Such damage will disrupt the passage of impulses from the brain to other parts of the body resulting in the emergence of a wide variety of neurological symptoms. There is no cure for MS, but a number of medications are capable of slowing the progression of MS and reducing the frequency and severity of relapses. Physical therapy, balanced diet, medications that provide symptomatic relieve of pain, spasms and G.I. disturbances may be used.

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1. Does the program meet the learning objectives?

Define MS & state its pathogenesis.	YES	NO
List types of MS.	YES	NO
Describe symptoms of MS.	YES	NO
State methods used to diagnose MS.	YES	NO
List medications used in treating MS.	YES	NO
Discuss side effects associated with MS treatments	YES	NO

2. Was the program independent & non-commercial

	YES	NO
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3. Relevance of topic

	Low Relevance		Very Relevant
	1 2 3	4 5 6	7
4. What did you like most about this lesson? _____
5. What did you like least about this lesson? _____

Please Mark the Correct Answer(s)

1. **MS occurs as a result of:**
 - A. Narrowing of cerebral blood vessels
 - B. Absence of electrical impulses in the spinal cord
 - C. Damage to the myelin
 - D. Absence of neurotransmitters in certain portions of the brain

2. **The most common type of MS, which affects about 85% of patients, is:**
 - A. Primary-Progressive MS B. Relapsing-Remitting MS
 - C. Progressive-Relapsing MS D. Secondary-Progressive MS

3. **Which of the following is not considered a risk factor for MS?**
 - A. Mental disorders
 - B. Geographic & environmental factors
 - C. Infection
 - D. Presence of immune-mediated response

4. **Which of the following is not a common symptom of MS?**
 - A. Muscle spasticity B. Vision impairment
 - C. Ataxia D. Mood elevation

5. **Limited vision caused by involuntary eye movements is termed:**
 - A. Dysphonia
 - B. Dysarthria
 - C. Nystagmus
 - D. Dysphagia

6. **Which statement is false about MS?**
 - A. Characterized by presence of plaques in the brain
 - B. Strikes persons of all ages
 - C. Does not affect the peripheral nervous system
 - D. MS is a curable disease

7. **The presence of plaques in the brain may be confirmed by the use of:**
 - A. Magnetic Resonance Imaging B. Blood tests
 - C. Lumbar puncture D. Nerve reflexes

8. **Which of the following drugs is considered an immunomodulator?**
 - A. Mitoxantrone B. Methyl prednisolone
 - C. Dimethyl fumarate D. Glatiramer

9. **Which drug is administered by IV infusion every 4 weeks for treating MS?**
 - A. Interferon beta-1b
 - B. Natalizumab
 - C. Teriflunomide
 - D. Fingolimod

10. **Progressive multifocal leukoencephalopathy (PML) is an adverse effect that may occur with:**
 - A. Interferon beta-1a
 - B. Mitoxantrone
 - C. Natalizumab
 - D. Dimethyl fumarate

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