Multiple sclerosis (MS) is a remitting-relapsing inflammatory demyelinating disease of the central nervous system in which axonal damage is often seen. The hallmark of the disease is the involvement of separate regions of the brain and spinal cord at different time points. The deficits appear, persist for a few days to months, and then resolve spontaneously. The presentation can be very dramatic and lead to immediate suspicion of the diagnosis or can be very subtle, with the diagnosis uncertain for months or years.

The cause of the disease is unknown but a number of hypotheses have been advanced. One of these is that the disease is a chronic viral infection of the oligodendrocytes, the cells that manufacture myelin. No such viral agent has been consistently isolated despite great effort. Another hypothesis is that this is an autoimmune disease; however, although multiple reactivity against several myelin antigens has been observed, no unique antigen has been demonstrated. There is evidence that an environmental factor (possibly an infection) is associated with risk of disease, and genetic susceptibility appears to be a risk factor, but these have not pointed to any specific cause. Currently, the consensus is that among those with a genetic susceptibility, exposure to any 1 of a small group of infectious agents in early life may cause a subtle distortion in immune reactivity that later manifests as MS.

The pathogenesis of MS appears to involve an immunologic attack on myelin antigens. This attack is initiated by activated myelin-reactive T cells, which are able to pass through the intact blood-brain barrier. On reaction with their cognate antigens, these cells initiate an inflammatory process that locally abolishes the blood-brain barrier and allows passage of other immune-active cellular and humoral factors that specifically damage myelin and secondarily impair axonal function. The resulting area of inflammatory demyelination (“plaque”) results in the characteristic deficits of the disease. Not all plaques result in symptoms, however. As the plaque evolves the inflammation decreases and scarring by astrocytes (astrocytosis or gliosis) results. Magnetic resonance imaging (MRI) of the brain and spinal cord can demonstrate these plaques, which have characteristic shapes and locations in the brain and spinal cord. This type of imaging is useful in making the diagnosis. Active plaques with ongoing inflammation typically will show enhancement on T1-weighted MRI images on injection of gadolinium contrast. This enhancement of active lesions has been used as a surrogate measure of disease activity in clinical trials.

Four types of disease course are currently recognized [1]. Relapsing-remitting MS, the most common type, consists of relapses and remissions (which are not always back to baseline). Secondary progressive MS consists of relapses followed by a steady progression of deficit that may be punctuated by further relapses. Primary progressive MS (about 15% of cases) is steadily progressive from onset and is usually seen in older patients. Progressive-relapsing MS (rare) begins with slowly progressive deficits upon which occasional relapses and remissions are superimposed. Benign MS (probably less than 10% of cases) is a form of relapsing-remitting MS in which there are no significant residual deficits 15 years from onset. At 25 years from onset, approximately 90% of initially relapsing-remitting MS patients enter the secondary progressive stage [2]. Finally, acute malignant MS has rapidly developing severe deficits that leave the patient bedbound and helpless within 1 or 2 years from onset.

In about two thirds of cases, the disease begins with discrete neurologic deficits that reflect acute focal demyelinating lesions. These include unilateral visual loss, diplopia, dysarthria, dysphagia, weakness or numbness of the hands or feet, hemiparesis, hemisensory loss, gait ataxia, and bladder or bowel dysfunction. Generally, the deficits remit in a few days to weeks to be followed by a relapse months to years later in another part of the central nervous system. The remissions may not always be complete, and often there is accumulation of deficits. This accumulation of deficit results in roughly 50% of patients having difficulty with ambulation 15 years after onset of the disease [3].

The disease affects women more often than men (1.5 to 2:1), usually in the teens or 20s, although the disease has been described in children and the elderly. The disease is not uniformly distributed in the world, being common in Europe, Scandinavia, Britain, Canada, and the United States, and rare in Africa, India, and Asia. The disease is more common in particular ethnic groups and in those with particular HLA haplotypes, being more common among whites and rare

From the Division of Neuroimmunology, Department of Neurology, Wayne State University School of Medicine, Detroit, MI.
among blacks and Asian persons. There is a north-south gradient in the prevalence of the disease, with the disease more common in regions further away from the equator. In the United States, the prevalence of the disease is between 20 and 100/100,000 and is more common in the northern latitudes.

Numerous clinical trials of various agents to decrease the underlying disease activity have been published. MS clinical trials are very challenging to design and carry out because there are no sensitive or unique markers of disease activity, which is highly variable and unpredictable. Furthermore, there is a strong placebo effect in this disease. Outcome variables measured in studies include frequency and severity of relapses (defined in a standard way, eg, a transient increased score on the Extended Disability Status Scale [EDSS, Table 1]) [4], proportion of patients without relapses, proportion of patients progressing, and MRI activity as defined by new or enlarging lesions on T2-weighted images or the number of enhancing lesions on T1-weighted images.

The treatment of MS involves several dimensions: prevention of relapses and progression by immunomodulatory agents, treatment of individual relapses with corticosteroids, and treatment of residual symptoms and disability. The former are discussed in detail below. A comprehensive review of MS was recently published in the *New England Journal of Medicine* [3].

**CASE STUDY**

**Initial Presentation**

A 24-year-old female college student presents with blurring of vision in the right eye and a periorbital headache made worse by eye movement.

**History and Physical Examination**

The patient has a previous history of unsteady gait of 1 week’s duration that occurred 1 year ago. She also experienced double vision lasting 2 or 3 days about 2 years ago.

Neurologic examination shows a visual acuity of 20/70 in the right eye with an afferent papillary defect, a trace right central facial palsy, left hyperreflexia, and an upgoing left toe. Otherwise, history and physical examination are unremarkable.

**Laboratory Evaluation**

An MRI of the brain shows several areas of increased signal on T2-weighted images; they are scattered in the centrum semiovale and with an ovoid shape, with the major axis pointing towards the ventricles. Two of the lesions abutted the ventricle and 1 of them enhanced with gadolinium contrast injection. The cerebrospinal fluid (CSF) contains 12 lymphocytes. The CSF protein and glucose are normal. Four oligoclonal bands are present in the CSF; none are present in the serum. Other workup includes measurement of serum vitamin B₁₂, rapid plasmin reagin, and Lyme titers and tests for human T-cell leukemia/lymphoma virus (HTLV), HIV, and antinuclear antibody, all of which are normal or negative.

**What is the differential diagnosis of the optic neuropathy in this patient?**

The differential diagnosis of the patient’s illness consists of those diseases that can cause optic neuropathy as well as diseases that cause white matter lesions. These include multiple sclerosis, syphilis, vitamin B₁₂ deficiency, Lyme disease, sarcoidosis, and systemic lupus erythematosus. Rarely, HIV disease can mimic MS. Myelopathy can be caused by the entities listed for the differential diagnosis of optic neuropathy as well as by HTLV-1 infection. Most of these are quite uncommon and can easily be eliminated by simple tests.

**How is the diagnosis of MS made in this case?**

The diagnosis of multiple sclerosis depends on the demonstration of multiple areas of demyelination in the central nervous system, occurring at different time points, with or without evidence of intrathecal immune activation. The multiple lesions can be documented by clinical neurologic examination, MRI, and evoked potentials. Intrathecal immune activation is demonstrated by the presence of oligoclonal banding in the CSF, with no corresponding oligoclonal bands in the serum. A list of the criteria for the diagnosis of multiple sclerosis is given in Table 2.

This patient clearly has clinically definite multiple sclerosis, with the current episode of optic neuritis following previous episodes of gait ataxia and diplopia, the latter consistent with a brain stem lesion. Both of these previous episodes

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**Table 1.** Extended Disability Status Scale

<table>
<thead>
<tr>
<th>Score</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal</td>
</tr>
<tr>
<td>4</td>
<td>Ambulates &gt; 300 meters without aid</td>
</tr>
<tr>
<td>5</td>
<td>Limited ambulation without aid</td>
</tr>
<tr>
<td>6</td>
<td>Ambulates with aid</td>
</tr>
<tr>
<td>7</td>
<td>Essentially restricted to wheelchair</td>
</tr>
<tr>
<td>8</td>
<td>Essentially restricted to bed</td>
</tr>
<tr>
<td>9</td>
<td>Restricted to bed and helpless</td>
</tr>
<tr>
<td>10</td>
<td>Death due to multiple sclerosis</td>
</tr>
</tbody>
</table>

resolved. There is evidence of previous lesions on the neurologic examination, ie, the left hyperreflexia and the right facial palsy. The MRI and CSF findings are strongly supportive of the diagnosis. Other potential causes of this patient’s illness have been ruled out by the workup.

- How should the patient’s exacerbation be treated?

**Corticosteroids**

The acute relapse is treated with high-dose corticosteroids. As a result of small studies and case reports published in the 1950s and 1960s, adrenocorticotrophic hormone (ACTH) was subjected to a proper clinical trial as a treatment for MS exacerbations, in which patients were randomized to ACTH or placebo. Benefit was observed for the ACTH group compared with the placebo group, although the final outcome was the same at 4 weeks, suggesting that the ACTH accelerated the rate but not the degree of recovery [5]. The observation of the efficacy of high-dose methylprednisolone in other inflammatory diseases, such as lupus nephritis, glomerulonephritis, and acute renal transplant rejection, stimulated open-label exploratory studies of its use in MS [6,7]. These were followed by double-blind, placebo-controlled studies, which elucidated the superiority of high-dose intravenous methylprednisolone over placebo in acute attacks of MS [8,9]. Finally, double-blind, controlled trials comparing the efficacy of ACTH to that of methylprednisolone showed that the 2 were equivalent [10,11]. However, methylprednisolone is more convenient to use than ACTH (which is no longer available), and is now almost exclusively used. There is currently no standard dose of methylprednisolone, although most neurologists will give 1 g per day for 3 to 7 days.

The first step in managing this patient is to treat the optic neuritis. Therapy of optic neuritis used to consist of oral prednisone, intravenous methylprednisolone, or no therapy at all since in most cases the disease resolved spontaneously. In 1992, however, these 3 options were tested in a randomized, placebo-controlled trial. In that study of acute optic neuritis, 457 patients with were randomized to receive either oral prednisone (1 mg/kg per day for 14 days), intravenous methylprednisolone (1 g daily for 3 days, followed by 11 days of oral prednisone), or oral placebo [12]. The principal outcome variables were visual acuity, visual fields, color vision, and contrast sensitivity measured at baseline, at 4, 15, and 30 days, at 7, 13, and 19 weeks, and at 6 and 12 months. Significant differences in the visual outcomes were noted at days 4 and 15 in favor of the methylprednisolone arm. Outcome in the oral prednisone group did not differ from that in the placebo group. At 6 months, the methylprednisolone group was still slightly better off than the other 2 groups in all the outcome variables except for visual acuity. It was found that patients on the oral prednisone arm had a slightly greater risk of recurrent relapse as compared with the other 2 arms, and oral prednisone is no longer given for optic neuritis.

**Initial Treatment**

The patient is treated with intravenous methylprednisolone 1 g per day for 5 days and then put on an oral prednisone taper. Her visual acuity returns to normal by the fourth day of treatment. The patient elects to delay starting immunomodulatory medications.

**Four Months Later**

Four months after the optic neuritis episode, the patient develops double vision and numbness of the feet. This progresses proximally over 1 week, so that she is numb from the waist to the waist.
down. She has some urinary urgency but no incontinence. Examination reveals a mild left internuclear ophthalmoplegia, decreased sensitivity to pinprick below the T10 spinal level, and bilaterally upgoing toes. The patient is clearly having a relapse of her MS, with 2 separate lesions occurring simultaneously. One lesion is in the brain stem, affecting the medial longitudinal fasciculus and causing the double vision; the other lesion is in the thoracic spinal cord, causing the numbness below the waist. The patient is again treated with pulsed methylprednisolone, 1 g per day intravenously for 5 days, with resolution of the diplopia and bladder difficulties but with a slight numbness remaining. She is urged to start immunomodulatory therapy.

- What agents are available for long-term management of MS?

**Immunomodulating Agents**

Long-term management involves the use of various immunomodulating agents. These drugs are used for their action in decreasing the activity of the disease and are not useful for the treatment of individual relapses. The precise actions of these drugs are not clear, but complex immunomodulatory activities, although not immunosuppression, are involved. Three immunomodulating agents are approved for the treatment of MS in the United States.

**The Interferon Betas**

Interferon beta-1b. The original rationale behind the use of IFNb-1b (Betaseron, Berlex Laboratories) was that it would inhibit a putative etiologic viral agent [13]. In the pivotal trial, which led to the licensing of the product, 372 ambulatory patients who had had at least 2 relapses in the 2 years before entering the trial were randomized to receive IFNb-1b 250 µg, 50 µg, or placebo subcutaneously every other day [14]. The main outcome variable was relapse rate. The rate of new lesions on MRI and accumulation of deficit were secondary outcomes. The annualized relapse rates were 1.27, 1.17, and 0.84 in the placebo, low-dose, and high-dose groups, respectively; the differences were statistically significant. There was no difference in the accumulation of disability (as measured by the EDSS) among groups, although the study was not powered to detect this. In a companion study, MRI activity of the disease was defined as new, recurrent, or enlarging areas of increased signal intensity on T2-weighted images [15]. It was found that the high-dose group had an 80% decrease in MRI activity compared with the placebo group. Another study looked at the effect of IFNb-1b on the frequency of enhancing lesions, which is a measure of the degree of breakdown of the blood-brain barrier. In this study, the frequency of lesion enhancement before treatment (for a period of several months) was compared to that after treatment (for 6 months) in 14 patients. The average number of enhancing lesions was reduced from 3.06/month before treatment to 0.48/month after treatment [16].

Interferon beta-1a. The pivotal trial involved 301 patients with early disease who were randomized to either placebo or IFNb-1a (Avonex, Biogen) 30 µg intramuscularly once per week [17]. The primary outcome variable was time to sustained progression of MS, defined as a 1.0 increase on the EDSS sustained for 6 months. The study was stopped early as the dropout rate was smaller than anticipated. At 2 years, there were 172 patients in the study. The proportion of patients progressing by the end of 104 weeks was 21.9% in the treatment group versus 34.9% in the placebo group. In a separate analysis, decrease in disability progression was not statistically significantly at 52 weeks but became so at 78 weeks [18]. At 1 year, relapse rates were not different between groups [19], but at 2 years the annual relapse rate was 0.61 in the IFNb-1a group compared with 0.9 in the placebo group, a statistically significant difference. The number of enhancing lesions also decreased in the treatment group compared with placebo; on T2-weighted images, there was less accumulation of lesions in the treatment group in the first year, but no difference in the second year [17].

**Dosing**. The optimal dosing of the interferons for MS has not been completely settled at this time. In the Prevention of Relapses and Disability by Interferon beta-1a Subcutaneously in Multiple Sclerosis (PRISMS) study, 560 ambulatory patients with remitting-relapsing MS were randomized to either placebo, low-dose IFNb-1a (22 µg 3 times per week subcutaneously), or high-dose IFNb-1a (44 µg 3 times per week) [20]. The patients were evaluated for relapse rate, accumulation of deficit, and MRI activity (as measured by accumulation of lesions on T2-weighted scans and number of enhancing lesions). The treatment groups showed benefit on all measures with a trend toward a dose-response, although formal statistical analysis was limited to comparison of each of the treatment groups to placebo. In the Once Weekly Interferon for MS (OWIMS) study, 293 remitting-relapsing patients with mild disability were randomized to receive either placebo, IFNb-1a 22 µg, or IFNb-1a 44 µg subcutaneously once per week [21]. The primary outcome measures were number of lesions on T2-weighted MRI scans and number of enhancing lesions on T1-weighted scans, which were combined into “combined unique” MRI lesions. Secondary outcomes were clinical measure (relapse rate, time to first relapse, severity of relapse, and use of corticosteroids for relapses). There was a significant reduction in combined
unique lesions in the treatment groups, with a dose-response effect. There was no clear effect on the clinical variables. The preparation of IFNb-1a used in these studies was Rebif, which is licensed for use in Europe and Canada. The authors of the study did a meta-analysis of previous studies of IFNb-1a and showed an overall trend to dose-responsiveness of the disease. It should be noted that a strict comparison of the dosages of the various preparations of the interferon betas is difficult, since different methods of titration are used, different dosages are given with different frequencies and different potencies, with different routes of administration and different relative absorptions.

Side effects. The main side effects of the interferon betas are a flu-like syndrome of malaise, myalgias, and possibly fever as well as injection-site reactions. This can be prevented by premedication with acetaminophen or ibuprofen before the injection, which is taken at night. Many neurologists initiate therapy at low doses and gradually titrate up to the full dose over several weeks. A typical regimen is 1/4 dose for 2 weeks, 1/2 dose for 2 weeks, and 3/4 dose for 2 weeks, followed by the full dose thereafter. Depression is occasionally noted. Leukopenia and mild elevations of liver enzymes are noted but are not usually severe. Complete blood counts and liver function tests are obtained at baseline and every 3 months thereafter. The drugs have caused miscarriages in animal studies, and although there are no human data concerning teratogenicity, the drugs should not be used in pregnancy, or in patients intending to become pregnant soon. The drugs should be stopped at least 1 or 2 menstrual cycles before pregnancy is attempted.

Glatiramer Acetate
The third immunomodulating agent available is glatiramer acetate (Copaxone, Teva Pharmaceuticals), a mixture of 4 synthetic oligopeptides consisting of alanine, glutamate, lysine, and tyrosine. The exact mode of action of this agent is unknown, but it is thought to cross-react with myelin basic protein, an important constituent of myelin, and may inhibit cell-mediated immune responses to this protein. In the pivotal study examining the effect of glatiramer on MS, 251 ambulatory patients with relapsing-remitting MS were randomized to placebo or glatiramer 20 mg subcutaneously every day and followed for 24 months [22]. The main outcome variable was relapse rate. The glatiramer group had an annualized relapse rate of 0.59 compared with 0.84 in the placebo group. Patients on glatiramer were more likely to have no change in disability or improvement than patients on placebo. An extension study that followed the subjects beyond the 24 months showed sustained effect of the drug [23]. An MRI study of 10 patients showed a decline in MRI activity (defined as new enhancing lesions) of 57% from baseline activity [24]. A larger, placebo-controlled study of the effect of glatiramer acetate on MRI activity showed a sustained 35% reduction in the number of gadolinium-enhancing lesions in the glatiramer group compared with the placebo group in a short study of 9 months' duration [25]. The clinical efficacy of the drug was demonstrated to be sustained for at least 6 years in an open label study of glatiramer acetate involving 152 of the 251 patients in the original pivotal study [26]. An advantage of glatiramer acetate is that no blood monitoring is required.

The main side effect of glatiramer acetate is a reaction consisting of flushing, chest tightness, and tachycardia, beginning a few minutes after the injection. The reaction is self-limiting. Occasionally there is redness, swelling, and itching at the injection site. This is annoying but self-limiting.

Choosing Among Agents
No placebo-controlled head-to-head comparisons of the 3 immunomodulating agents have been done. However, in a prospective open-label study, 156 remitting-relapsing ambulatory MS patients were followed after being allowed to choose their treatment (no treatment, Avonex 30 µg intramuscularly once per week, Betaseron 250 µg subcutaneously every other day, or Copaxone 20 mg subcutaneously every day) [27]. The patients in the 4 groups had similar EDSS scores and rates of relapse at baseline. Compared to no treatment, after 12 months of therapy relapse rates in patients taking Betaseron (37%) and Copaxone (36%) were significantly reduced in contrast to patients taking Avonex (12%), who did not demonstrate a significant reduction.

The decision of which drug to use is not simple, and is dictated in part by risk of side effects, efficacy, and convenience. All 3 drugs are effective. INFb-1b is quite effective, but side effects have to be managed carefully. INFb-1a is much more convenient to use, but issues of dosing and relative efficacy arise. Glatiramer acetate has the fewest side effects and its efficacy appears to be sustained for at least 6 years.

Further Treatment and Course
The patient agrees to start immunomodulatory therapy with glatiramer acetate, which she has no difficulty with. She continues in college and graduates with a degree in business administration. She after, she obtains a job as an administrator in a software company and gets married. Six months after starting the glatiramer acetate, she has an episode of numbness in both arms but decides to forgo corticosteroids, as the episode is very mild and does not interfere with her usual activities. After 2 years of marriage, she wishes to become pregnant.
Pregnancy and MS

There is no reason for an otherwise healthy MS patient to avoid pregnancy, so long as the infant can be cared for and the patient understands that while the risk of relapse during pregnancy is decreased, there is a temporary rebound in risk of relapse after pregnancy. In the most recent study of pregnant MS patients, there was a postpartum rebound in risk of relapse in the 3 months after childbirth, with a subsequent decrease in risk to baseline [28]. In other studies of women with MS who subsequently became pregnant, it was found that the risk of an MS relapse was not increased during pregnancy, nor was the risk increased in a 6-month period after pregnancy [29,30].

There is no adequate systematic study of the effect of the immunomodulatory agents on pregnancy or the fetus. None of the immunomodulatory agents is recommended for use during pregnancy, and therefore, all such drugs should be discontinued before pregnancy is attempted.

Further Course

The patient discontinues her glatiramer and becomes pregnant shortly thereafter. Her pregnancy and labor are uncomplicated, and she gives birth to a healthy baby. She elects to breast-feed and does not resume the glatiramer during the postpartum period. Six weeks after the birth, she develops left-sided periorbital pain and slight dimming of vision, which lasts a week and spontaneously resolves. She decides against therapy with corticosteroids. She resumes her glatiramer acetate 6 months later, after the baby has been weaned.

Over the next 4 years, the patient has occasional mild relapses, which are treated with intravenous methylprednisolone when severe enough to compromise her function, but this is unusual. During the past 2 years, however, she has noted a slow deterioration of her gait, with gradually more frequent stumbling and the occasional need to use a cane for extended walks, especially during trips to the shopping mall. On examination, the tone in the legs is increased, there is mild weakness in the legs, and the reflexes are brisk, with bilaterally upgoing toes. Vibration sense in the toes is decreased bilaterally. There is no spinal sensory level to pinprick.

What is the significance of this patient’s chronic progressive myelopathy?

In the absence of any other cause, such as B12 deficiency, lupus, syphilis, HTLV-associated myelopathy, or HIV disease, the diagnosis is secondary progressive multiple sclerosis.

Management of Secondary Progressive MS

Since there is currently no relapse, pulsed corticosteroids are not indicated. The question of the effect of immunomodulating agents on the progressive aspect of the disease was examined only recently, in a European study [31]. In this study, 718 patients with secondary progressive MS were randomized to IFNb-1b 4 million international units (MIU) subcutaneously every other day for 2 weeks, followed by 8 MIU every other day thereafter, or placebo, and were followed for 2 to 3 years. The primary outcome was a sustained increase in the EDSS score by 1.0 step (if baseline EDSS were less than 6; 0.5 step if baseline EDSS was 6.0 to 6.5) for at least 3 months. The study was stopped early because an interim analysis showed clear evidence of benefit of the drug. It was found that there was a highly statistically significant difference in progression rates favoring the treatment group compared with the placebo group. The treatment group had progression delayed for 9 to 12 months compared with controls. Benefits were also seen for time to becoming wheelchair-bound, frequency and severity of relapses, and on MRI activity, all favoring the treatment group over the placebo group. However, in a similar study done in North America, there appeared to be no decrease in the rate of progression [32]. The reason for these differing results is unclear at this time, but probably involves the fact that the patients in the 2 studies were not completely comparable. The patients in the North American study had a higher baseline disability than those in the European study, and the European study patients had a higher rate of relapses than the North American patients, which suggests that those with relatively lower levels of disability along with clinical evidence of relapsing inflammatory activity are more likely to respond to immunomodulating agents than a population with higher disability and fewer relapses. Since there is therefore some evidence of efficacy, the case study patient should be offered treatment with IFNb-1b.

More recently, the FDA approved the use of mitoxantrone, an anthracenedione, for secondary progressive MS. The phase III pivotal trial, the Mitoxantrone in Multiple Sclerosis Study (MIMS), recruited 194 patients with secondary progressive or relapsing progressive MS and randomized them into 3 arms: placebo, low-dose mitoxantrone (5 mg/m² of body surface area intravenously every 3 months), and high-dose mitoxantrone (12 mg/m² intravenously every 3 months). Patients on the 2 doses of...
mitoxantrone benefited in all measures of disease activity, including reductions in relapse rate, progression of deficit, and MRI activity of disease. The total cumulative dose is limited to a maximum of 140 mg/m² by the potential cardiotoxicity of the drug [33].

Other options for secondary progressive MS include monthly intravenous infusions of methylprednisolone 1000 mg and monthly intravenous infusions of cyclophosphamide, although there are no definitive data on their use.

What are the other issues in the treatment of multiple sclerosis?

There are many issues and complications to be found in any chronic, unpredictable, potentially disabling, progressive disease that affects young adults just beginning their lives. Problems faced by MS patients include depression, spasticity, incoordination, cognitive difficulties, and bladder and bowel dysfunction. Each of these is treatable, with varying degrees of effectiveness. Sometimes referral to other specialists (psychiatrists, urologists, physiatrists) is necessary for optimal care.

Change in Therapy, Further Course

The patient decides to start therapy with INFβ-1b and discontinues glatiramer acetate. She has some mild myalgias after starting the INFβ-1b, but these are well-controlled with ibuprofen. Two years later, her condition has not changed very much and she is able to get around well, with occasional use of a cane when walking long distances, particularly at shopping malls.

References


