Stepped-Care Approach to Treating MS: A Managed Care Treatment Algorithm

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ABSTRACT

OBJECTIVE: To introduce a model treatment algorithm for use in the managed care setting as a strategy to provide ongoing disease management and long-term care for patients with multiple sclerosis (MS), with the goal of delaying disease progression and the associated disability and cognitive dysfunction.

SUMMARY: MS is a chronic inflammatory disorder of the central nervous system that is associated with progressive disability and cognitive dysfunction. Currently, management of MS involves planning an effective long-term treatment strategy that can delay the progression of the disease. This article reviews a typical stepped-care approach to treating MS that is based on the concept of a platform drug, which is an agent that provides baseline immunomodulatory action throughout the course of the disease.

Considerations for selecting a platform therapy include the effect on the full spectrum of MS (disability, relapses, lesion load, and atrophy as well as patient compliance and the potential impact of neutralizing antibodies [NAbs]). Currently, 4 first-line therapies are approved for relapsing MS: the 3 interferon beta (IFNβ) products and glatiramer acetate. Of these, the IFNβs are generally recommended as platform therapy because all have shown significant effects on relapses, magnetic resonance imaging parameters of the disease, and because intramuscular (IM) IFNβ-1a (Avonex) and subcutaneous (SC) IFNβ-1a (Rebif) have been shown to slow the progression of sustained disability.

Patients being treated with IFNβs can develop NAbs to the drug, which can lead to a loss of efficacy and subsequent occurrence of breakthrough disease. The 3 different formulations of IFNβ are associated with a varying incidence of NAbs (IM IFNβ-1a, 5%; SC IFNβ-1a, 24%; IFNβ-1b [Betaseron], 45%). Antibodies also form against glatiramer acetate, although their clinical significance needs to be elucidated. As the disease progresses or has periods of aggressive activity, the stepped-care approach is to add other agents onto the platform therapy to improve control of the disease.

CONCLUSION: Stepped care, as outlined in this model treatment algorithm for the managed care setting, is an effective method to achieve the fundamental goal of MS treatment, that is, to delay disease progression and the associated disability and cognitive impairment.

KEYWORDS: Multiple sclerosis, Stepped care, Treatment algorithm

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Multiple sclerosis (MS) is a chronic demyelinating disease of the central nervous system. Natural history data suggest that in a majority of patients diagnosed with clinically definite MS, the disease progresses from an initial relapsing-remitting form to a secondary progressive type.[1] Delaying progression of the disease and the associated disability and cognitive dysfunction is one of the fundamental goals of MS therapy. To achieve this objective, an individualized, dynamic, long-term treatment strategy should be implemented along with ongoing monitoring of disease activity. The treatment plan should be able to adapt to the changing needs of the individual patient, based on clinical findings of disease progression, severity of MS symptoms, increase of disease burden on magnetic resonance imaging (MRI), and development of neutralizing antibodies (NAbs).

Approved first-line therapies for relapsing-remitting MS include the 3 interferon beta (IFNβ) products: intramuscular (IM) IFNβ-1a, (IM IFNβ-1a [Avonex, Biogen Idec Inc., Cambridge, MA]); subcutaneous (SC) IFNβ-1a (SC IFNβ-1a [Rebif, Serono, Inc., Rockland, MA]); and IFNβ-1b (Betaseron, Berlex Laboratories, Montville, NJ) and glatiramer acetate (Copaxone, Teva Neuroscience, Inc., Kansas City, MO).[2] Consequently, these drugs are used as baseline immunomodulatory agents (platform drugs) in the treatment of MS. These treatments are proven to slow various aspects of MS; however, most patients will experi-

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ence disease progression. In patients undergoing treatment with a platform drug, ongoing monitoring of disease activity, including regular MRI scans, may identify breakthrough disease (i.e., frequent exacerbations and increased disability). Once identified, clinicians can add corticosteroids or a number of other secondary agents, as necessary, to the platform drug to manage breakthrough disease.[3] This article reviews issues relating to long-term treatment strategies and ongoing disease management in MS and provides a model treatment algorithm for use in the managed care setting.

Diagnosis and Therapy Selection

Diagnosis

A single clinical event indicative of demyelination is often the earliest symptom detected in patients with MS. Typically, patients present to their primary care physician with an isolated clinical event, for example, optic neuritis in one eye or numbness on one side of the body. Once a patient is referred to a neurologist, a diagnosis of clinically isolated syndrome (CIS) is made based on a neurologic or ophthalmologic examination, or both, confirming the clinical event consistent with demyelination involving the optic nerve (optic neuritis), spinal cord (incomplete transverse myelitis), or brainstem or cerebellum (brainstem or cerebellar syndrome).[4]

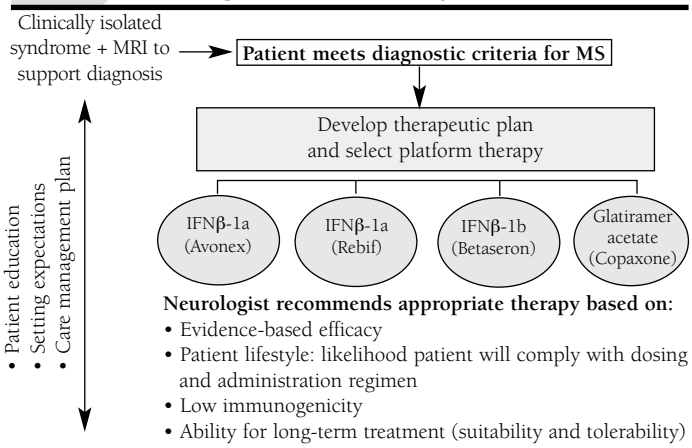
Following a diagnosis of CIS and exclusion of alternate diagnoses, the patient's risk of developing clinically definite MS (CDMS) is evaluated. Historically, a diagnosis of CDMS was made following the occurrence of a second clinical demyelinating event.[5] However, because the time between the first and second attacks varies considerably, diagnosis and therapy initiation could take several years. Many studies have therefore evaluated the risk of developing CDMS in patients diagnosed with CIS using paraclinical measures, such as MRI, evoked potentials, and examination of cerebrospinal fluid (CSF) for the presence of oligoclonal bands. Of these measures, MRI has been shown to be the most sensitive method for predicting the development of CDMS in patients with suspected MS.[6] Further, the prognostic value of MRI in MS has been demonstrated in prospective follow-up studies of patients with CIS.[7,8] Diagnostic criteria for MS now include MRI as a paraclinical diagnostic tool[9] because the presence of characteristic MS lesions on MRI is associated with a high risk of developing CDMS.[7,10,11]

Therapeutic Plan Development and Selection of a Platform Therapy

The National Multiple Sclerosis Society recommends initiation of treatment as soon as possible after a definitive diagnosis of MS is made and also recommends that treatment be initiated in patients at high risk of developing MS.[12]

Following the decision to initiate therapy, one of the first steps is selection of an appropriate platform drug (Figure 1), which is defined as an agent that can provide baseline immunomodulatory action throughout the course of the disease. Platform treatment may be adequate treatment for many patients for years; however, for patients with aggressive disease, additional agents can be added to

FIGURE 1 Diagnosis and Therapy Selection



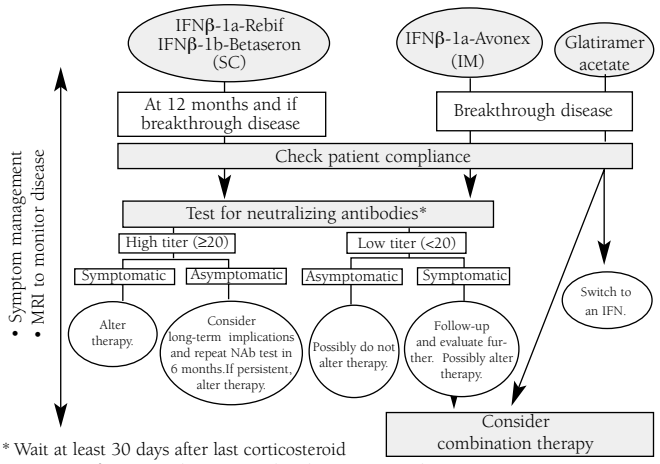
The decision to initiate treatment for multiple sclerosis (MS) is followed by careful consideration of the available first-line or "platform" therapies. Educating patients and setting realistic treatment expectations are also important factors in designing an effective long-term treatment plan. IFNβ = interferon beta; MRI = magnetic resonance imaging.

the platform drug, based on symptoms and disease progression. Platform therapy options are the 4 drugs that are approved by the U.S. Food and Drug Administration (FDA) for use in relapsing MS: IFNβ-1b, IM IFNβ-1a, SC IFNβ-1a, and glatiramer acetate. The relative efficacy, side effects, convenience, and compliance issues relating to these drugs (discussed in the article by William H. Stuart in this supplement) should be considered when evaluating the different platform drugs. IFNβs are recommended as platform therapy because they have an impact on relapses and lesions on MRI. In addition, IM and SC IFNβ-1a have been shown to slow the progression of sustained disability[13-16] and IM and SC IFNβ-1b therapies have been shown to significantly decrease brain atrophy.[17,18]

Given the long-term nature of MS treatment, the clinical aspects of the available platform drugs should be given careful consideration before initiating treatment. The complications that may arise due to the generation of NAbs to IFNβ also should be taken into account (for a detailed discussion, see the article by Howard S. Rossman in this supplement). These complications include reduced efficacy of the drug and cross-reactivity of NAbs that make switching between IFNβ products impractical. The neurologist must consider these factors and assist individual patients in selecting the appropriate agent rather than simply providing general information to patients and having them select a drug.

Ongoing patient education sets realistic expectations for agent effectiveness. For instance, after treatment initiation, a patient may experience exacerbations or relapses for some time. Generally, the IFNs require 3 to 6 months of treatment to become fully effective, and glatiramer acetate may take up to 9 months to become fully effective. Patients must also be aware of the potential side effects of the chosen platform therapy. Some of the common side effects associated with IFNβ treatment are injection-site reactions (mostly SC formulations), flu-like symptoms, and headache.[19-22] These side

FIGURE 2 Ongoing Disease Management



A dynamic treatment strategy consists of altering treatment as needed based on periodic monitoring of multiple sclerosis symptoms, disease burden on magnetic resonance imaging (MRI), patient compliance, and formation of neutralizing antibodies. IFNβ = interferon beta; IM = intramuscular; SC = subcutaneous.

effects can be managed, and appropriate patient education and ongoing monitoring can improve the experience and minimize the risk of patient noncompliance. Glatiramer acetate also is associated with injection-site reactions, including lipoatrophy.[23] Additional side effects with glatiramer acetate include chest pain, lymphadenopathy, and postinjection systemic reactions.[24]

Ongoing Disease Management

Effective, dynamic treatment strategies require initiation of platform therapy followed by regular, ongoing monitoring of patients for MS symptoms and disease activity. Ongoing monitoring of patients with MS can aid early detection of breakthrough disease. Occurrence of breakthrough disease is identified on an individual basis based on unacceptable disease progression. Possible criteria to assist in this determination include disability progression (e.g., increase of ≥1 point on the Expanded Disability Status Scale), multiple relapses in a short time span (e.g., ≥2 relapses in 6 months after 1 year of IFNβ therapy), development of new neurologic deficits, or deterioration evident on MRI.[3,25] Factors such as poor patient adherence and development of NAbs can contribute to the occurrence of breakthrough disease in patients on a platform drug and should therefore be monitored as well (Figure 2).

Symptom Management

The most common symptoms of MS are spasticity, fatigue, sexual dysfunction, bladder dysfunction, pain, and cognitive dysfunction. Other frequently noted symptoms include depression, bowel dysfunction, paroxysmal symptoms, and weakness.[26-33] Many MS symptoms can be interrelated such that one untreated symptom aggravates or leads to other symptoms, causing a cycle of interdependent symptoms. Disease progression also can lead to a wide range of complicating symptoms requiring additional treatments. Educating primary care physicians and nurses to identify symptoms that the patient is experiencing, and encouraging patients to avoid using multiple over-the-counter medications, vitamins, and herbal preparations are important symptom management tools. Not all drugs listed in the following section are approved by the FDA for use in MS. These drugs are discussed to educate pharmacists about medications that neurologists empirically have found useful and commonly prescribe for patients with MS.

Spasticity. Impairment of muscle function is one of the most common symptoms of MS, affecting an estimated 40% to 75% of patients,[28,34,35] and spasticity accounts for most of the physical disability seen in MS patients. Nonpharmacologic treatment options for spasticity include carefully planned, physician recommended exercise regimens (including aerobic exercise, stretching exercises to improve flexibility, and both active and passive movements that incorporate the full range of motion) and relaxation techniques (such as yoga, meditation, biofeedback, and tai chi). Pharmacologic treatments include baclofen ($GABA_B$-receptor stimulator [Lioresal]), tizanidine (α-adrenergic receptor agonist [Zanaflex]), and benzodiazepines.

Fatigue. Fatigue is reported by 80% to 97% of patients with MS and is characterized by a lack of energy, an overwhelming sense of tiredness, or a feeling of exhaustion.[36,37] Nonpharmacologic management of fatigue involves treating symptoms in patients that lead to fatigue, such as depression and sleep disturbances, and improving patient mobility through exercise. Pharmacologic treatments include the off-label use of modafinil (Provigil).[38,39] The *N*-methyl-D-aspartate antagonist amantadine (Symmetrel), methylphenidate (Ritalin), and amphetamines also are used off-label to treat fatigue.[29]

Depression. The lifetime prevalence of depression among patients with MS is 47% to 54%. Nonpharmacologic treatment consists of psychotherapy,[40-42] and pharmacologic agents used to treat depression include selective serotonin reuptake inhibitors (SSRIs [e.g., fluoxetine, sertraline, paroxetine, escitalopram, and citalopram]), tricyclic antidepressants (e.g., amitriptyline and nortriptyline), and atypical antidepressants (e.g., bupropion and venlafaxine).

Bladder dysfunction. Bladder symptoms are experienced by 80% to 96% of patients with MS and include overactive bladder (detrusor hyperreflexia) and urinary retention (overactive sphincter).[43] Treatment for overactive bladder consists of anticholinergics (e.g., oxybutynin and tolterodine), and treatment for urinary retention involves the off-label use of α-adrenergic antagonists (e.g., tamsulosin, doxazosin, and terazosin).[29,43]

Pain. Approximately 65% of patients with MS experience acute and subacute painful syndromes, the extent and impact of which are often underestimated.[44,45] Paroxysmal neuropathic pain is acute and intense; may worsen with age and disease progression[27]; and includes trigeminal neuralgia, which is triggered by sensory stimuli at various points on the face or head, Lhermitte's

phenomenon caused by cervical cord lesions, and dystonic spasms from paroxysmal dystonia. Treatment options for such pain include anticonvulsants, antispasmodics, and surgery. Constant neuropathic pain also can occur and may require the use of anticonvulsants, nonsteroidal anti-inflammatory drugs, opioid narcotics, nerve blocks, or tricyclic antidepressants.[44,45]

Sexual dysfunction. Approximately 48% to 75% of patients with MS may experience sexual dysfunction.[29] In men with MS, symptoms include erectile dysfunction, ejaculatory disorders, and difficulty achieving orgasm.[46,47] In women with MS, symptoms include reduced libido; reduced, altered, or painful sensations; reduced lubrication; difficulty achieving orgasm; and anxiety about incontinence.[48,49] Nonpharmacologic treatment options include addressing psychophysiologic issues that can contribute to sexual dysfunction; pharmacologic treatments include drugs for erectile dysfunction and lubricants. Discontinuation of SSRIs associated with sexual side effects also may be considered. Alternatives to SSRIs include tricyclic antidepressants and monoamine oxidase inhibitors.

Cognitive dysfunction. Impairment of cognitive processes is reported by 45% to 65% of patients with MS and is the symptom that is of greatest concern to patients.[50] Cognitive dysfunction most often includes impairment in learning and memory, attention, and information processing.[51] Nonpharmacologic cognitive rehabilitation is the main treatment option because, currently, no medications are approved for the treatment of cognitive impairment in MS.[52] Because the occurrence or progression of cognitive dysfunction is an indicator of active disease, treatment options for this symptom are the same as those used to delay the progression of the physical symptoms of MS (i.e., IFNβ and glatiramer acetate). IM IFNβ-1a has been shown to delay progression of cognitive dysfunction in patients with MS.[53]

Use of MRI to Monitor Disease Activity

Subclinical disease activity detected using MRI plays an important role in the longitudinal management of MS. Typically, lesions seen on MRI and used to assess disease activity include hyperintense lesions on T2-weighted images, hypointense lesions on T1-weighted images, and gadolinium-enhanced lesions on postcontrast images. Another measure that is being increasingly accepted as an important MS outcome is MRI measurement of CNS atrophy.[54] Increase in lesion load and progressive atrophy on MRI often may be clinically silent. Because brain MRI can detect disease activity that is subclinical, it is considered a more sensitive measure of disease activity than clinical findings.[55]

Generally, insurance coverage for MRI is available for the purpose of diagnosis but not necessarily for ongoing disease monitoring. Given that MRI measures can detect asymptomatic worsening of disease and thus help in making preemptive alterations to the treatment plan, it is recommended that MRI be performed periodically in patients with MS. Ideally, MRI should be performed every 12 months in patients with MS who are asymptomatic and more often (every 6 months) in patients who are symptomatic. Recommended MRI protocols are shown in Table 1.

TABLE 1 Recommended Magnetic Resonance Imaging Protocol

Brain, axial
- T1 noncontrast
- T1 postcontrast
- T2
- Fluid-attenuated inversion recovery (FLAIR)*

Brain, sagittal
- T1 noncontrast
- FLAIR

Spinal
- T1 sagittal
- T2 sagittal
- T2 axial
- Postcontrast (T1 axial, T1 sagittal)

**FLAIR is recommended to increase the sensitivity and specificity of hyperintense MS lesions.*

Testing for Neutralizing Antibodies

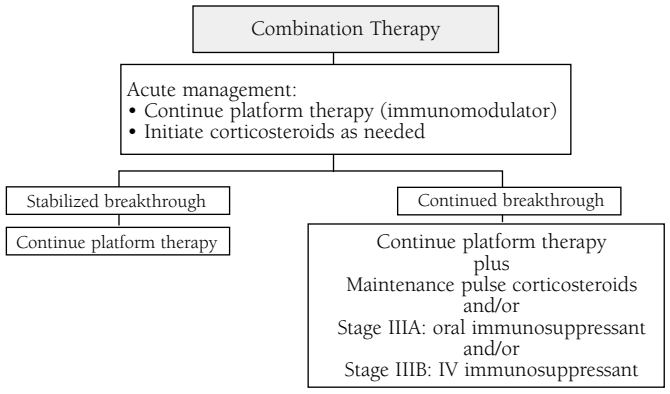
In patients with MS undergoing treatment with IFNβ, formation of NAbs to IFNβ can lead to a loss of efficacy of the drug and subsequent occurrence of breakthrough disease.[56,57] Antibodies also form against glatiramer acetate, although their clinical significance needs to be elucidated.[58] Early detection of NAbs through periodic testing can make the neurologist aware of the potential for recurrence of symptoms in patients who are NAb-positive.

The 3 different formulations of IFNβ are associated with varying incidences of NAbs: IM IFNβ-1a, 5%; SC IFNβ-1a, 24%; IFNb-1β, 45%. (For a detailed discussion, see the article by Howard S. Rossman in this supplement.) Consequently, the guidelines for NAb testing depend on which IFNβ product is being used as the platform drug. For patients using the more immunogenic IFNβ products (IFNβ-1b and SC IFNβ-1a), testing for NAbs should be done at 12 months or if breakthrough disease occurs. Patients who are being treated with the less immunogenic IM IFNβ-1a only need to be tested if breakthrough disease occurs.

The NAbFeron (IFNβ) antibody test (Athena Diagnostics) is the most commonly used, commercially available assay for NAbs. The cytopathic effect assay for NAbs, recommended by the World Health Organization, is based on the ability of NAbs in serum to interfere with the antiviral effects of IFNβ on human lung carcinoma cells.[59] NAbs are quantitatively expressed in neutralizing titers (a neutralizing titer is defined by a 50% inhibition of the activity of 10 IU/ml IFNβ). The threshold for NAb-positivity is defined by the presence in patient serum of NAb titers ≥20. The NAb titer appears to influence the persistence of NAbs. Patients with NAb titers >100 are more likely to remain NAb-positive for years. Patients treated with corticosteroids should not be tested for NAbs until 30 days after the last corticosteroid dose because corticosteroid treatment can temporarily suppress NAbs.

For symptomatic patients with high NAb titers (≥20), therapy alteration is recommended. For those with high titers who are asymptomatic, the NAb test may be repeated in 6 months. If NAbs are persistent, then therapy should be altered. For patients with low

FIGURE 3 Management of Breakthrough Disease Using Combination Therapy



Acute management of breakthrough disease involves the use of pulse corticosteroid therapy to stabilize the disease and continuing treatment with the first-line or "platform" drug. Continued breakthrough disease requires addition of either maintenance pulse corticosteroid therapy or a secondary agent to the platform drug.

titers of NAbs (<20) who are symptomatic, further evaluation and follow-up (including retesting in 6 months) should be considered before making any alterations to therapy. For asymptomatic patients with low NAb titers, no alteration to therapy is needed.

One of the concerns regarding testing for NAbs is expense. The cost of the NAbFeron test is estimated at $600.[59] However, given that yearly costs of IFNβ therapy may exceed $15,000, the benefit of identifying NAb-positive patients and switching them to alternate treatments is likely to be economically viable in the long term.[59]

Management of Breakthrough Disease

In general, for the purpose of designing a treatment plan, the MS disease process can be categorized into 3 stages. Stage I is the early part of the disease, Stage II involves acute breakthrough disease on treatment, and Stage III is characterized by continued breakthrough disease despite treatment. Depending on their response to therapy, and disease fluctuations and progression, patients may move from one stage to another and back.

Acute Breakthrough Disease

Management of breakthrough disease in Stage II involves the use of pulse corticosteroids. Typically, intravenous (IV) methylprednisolone 1 g/day is administered over 1 to 4 hours for 3 to 5 days.[60] The platform drug is continued during the management of breakthrough disease.

Continued Breakthrough Disease and Combination Therapy

Options available for the management of continued breakthrough disease in Stage III are switching from one platform drug to another, changing the dose of the current platform drug, and initiating combination therapy (Figure 3). No controlled studies have assessed the benefits of switching or of increasing the dose of the platform drug in patients with breakthrough disease. Further, increasing the dose of IFNβ or the frequency of administration may lead to increased incidences of adverse events and NAbs.[61-64] In addition, in patients who develop NAbs to IFNβ, switching between the 3 IFNβ formulations is not feasible because of the cross-reactivity between antibodies to the different IFNβ products.[61] Thus, the available treatment options for this chronic disease are reduced.

Combination therapy (i.e., addition of another agent to the platform drug) is the most effective way of managing continued breakthrough disease. An ideal agent for combination therapy is one that has biologic activity in MS with a mechanism of action that differs from the platform drug, provides synergistic efficacy, and has a low likelihood of additive toxicity.[25] Initially, maintenance pulse corticosteroids are added to the platform drug to stabilize breakthrough disease. In patients who require additional therapy, corticosteroids are followed by immunosuppressants or cytotoxic agents. Oral cytotoxic agents (eg, methotrexate, azathioprine, or mycophenolate mofetil) should be tried first (Stage IIIA) with IV cytotoxic agents (e.g., mitoxantrone and cyclophosphamide) being used as necessary (Stage IIIB). With the exception of mitoxantrone, which is approved for use in MS, these agents are approved for use in other diseases and are used off-label in the treatment of MS.[3] Often, a physician's experience with an agent and the cost of the drug influences the choice of agent when designing a combination therapy regimen.[25]

A number of novel immunomodulatory agents also are under investigation for the treatment of MS, mostly in phase I studies (Table 2). Phase II clinical trials have been reported with some agents, including the nonpeptide chemokine receptor antagonist, BX-471, and the humanized monoclonal antibody to $\alpha_4\beta_1$-integrin, natalizumab.[65,66] Natalizumab is currently being studied in phase III trials.

Conclusions

One of the fundamental treatment goals in MS is to delay the progression of disease and the associated disability and cognitive impairment. A stepped-care approach is an effective method for achieving this treatment objective and consists of the following: (a) initiating therapy with a platform drug in patients diagnosed with CDMS or patients with a CIS and at high risk of developing CDMS; (b) monitoring disease progression by assessing the severity of MS symptoms, noting the presence and number of lesions on MRI, and testing for NAbs; (c) identifying breakthrough disease early based on ongoing monitoring of disease activity; and (d) managing breakthrough disease.

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