



Belimumab (Benlysta)

A Breakthrough Therapy for Systemic Lupus Erythematosus

Raymond Lamore III, PharmD; Sapna Parmar, PharmD; Khilna Patel, PharmD Candidate; and Olga Hilas, PharmD, MPH, BCPS, CGP

INTRODUCTION

Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease that affects approximately 250,000 Americans, with a female-to-male ratio of 10:1.¹ SLE occurs predominantly in persons 15 to 64 years of age, and it is more common in women of African-American and Hispanic descent.² Suspected causes of SLE include failure of the immune system's regulatory mechanisms, immune complex deposition, and direct antibody-mediated cytotoxicity.³ This chronic, multifaceted inflammatory disease consists of a relapsing–remitting course with a broad range of possible clinical and serological manifestations.^{3,4}

Genetics, race, hormones, and the environment are risk factors for the development of SLE. Patients typically present with an array of symptoms, including fatigue, fever, arthralgia, and weight changes.³ As the disease progresses, symptoms may manifest in almost every organ system. The heart, lungs, kidneys, and brain are the organs most affected.⁵

Dr. Lamore is a pharmacy practice resident at The Johns Hopkins Hospital in Baltimore, Md. Dr. Parmar is a pharmacy practice resident at Stony Brook University Medical Center in Stony Brook, N.Y. Ms. Patel is a Doctor of Pharmacy Candidate for the year 2012 at St. John's University College of Pharmacy and Allied Health Professions in Queens, N.Y. Dr. Hilas is an Associate Clinical Professor of Clinical Pharmacy Practice at St. John's University and a Clinical Pharmacy Manager of Internal Medicine/Geriatrics at New York–Presbyterian Hospital/Weill Cornell Medical Center in New York, N.Y. Drug Forecast is a regular column coordinated by Alan Caspi, PhD, PharmD, MBA, President of Caspi & Associates in New York, N.Y.

In addition to the patient's clinical presentation, laboratory tests, such as a complete blood count (CBC) and a comprehensive metabolic profile, may provide diagnostically useful information when SLE is suspected. In advanced cases, diagnostic imaging, such as radiographs of involved joints, renal ultrasonography, chest radiography, and echocardiography, may be necessary, along with biopsy. Autoantibodies, such as antinuclear antibodies (ANAs), antiphospholipid antibodies, antibodies to double-stranded DNA (dsDNA), and anti-Smith (Sm) antibodies, are routinely assayed.⁵ Clinicians commonly follow the diagnostic guidelines published by the American College of Rheumatology (ACR); these recommendations were initially developed to standardize entry criteria for clinical trials.⁴

Various indices are available for determining disease status in patients with SLE.^{5,6} The most commonly used measure is the SLE Disease Activity Index (SLEDAI). A modification that has been made to SLEDAI over the years is known as the SELENA–SLEDAI. This modified index is a global composite index that includes 24 clinical and laboratory variables; a weighted score is given to each disease symptom.^{5–7}

Petri et al. suggested the following definitions of treatment outcomes based on changes in the SLEDAI index:⁷

- A reduction in the SLEDAI score of more than 3 indicates improvement.
- A change in the SLEDAI score of 3 indicates persistently active disease.
- A SLEDAI score of 0 indicates disease remission.

Disease activity categories were defined on the basis of SLEDAI scores, as follows: 0 = no disease; 1 to 5 = mild disease activity; 6 to 10 = moderate activity; 11 to 19 = high activity; and 20 = very high activity.⁷ Therefore, higher scores on the

SLEDAI represent increased disease activity.⁸

By contrast, the British Isles Lupus Assessment Group Index (BILAG) evaluates eight organ systems; each system is weighted by disease severity.⁶ BILAG consists of letter scores ranging from A to E, with A indicating the most severe stage of disease requiring treatment. Current clinical trials use BILAG scores of A and B.

In addition to SLE, three other major types of lupus have been identified:^{2,4,9}

1. Discoid lupus erythematosus (DLE) affects only the skin, causing thick, red, scaly rashes on the face, neck, and scalp.

2. Drug-induced lupus erythematosus (DIL), a rare form of lupus, is most commonly associated with hydralazine, (e.g., Apresoline, Novartis), which is used to treat hypertension, and procainamide (e.g., Pronestyl, Bristol-Myers Squibb), which is used to treat heart disease.

3. Neonatal lupus erythematosus (NLE) results when a mother's antibodies are transferred to her child at birth. The child may develop a rash, anemia, and potentially fatal heart problems.

Because SLE can be both debilitating and life-threatening, the disease can significantly affect quality of life. Patients with SLE experience increased fatigue, pain, rash, fever, abdominal discomfort, headache, or dizziness, which can present at the onset of a lupus-associated flare.² Life expectancy depends on the extent of organ damage at the onset of treatment. An early diagnosis can improve the prognosis. The 20-year survival rate for patients with SLE is 70%.¹⁰ If the disease is left untreated, the brain, heart, lungs, kidneys, and other major organs may be compromised.^{1,2,5}

There is no cure for SLE; medical treatment is aimed at reducing symptoms. Lifestyle modifications, such as exercise, smoking cessation, and appropri-

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ate nutrition, may also play a part in controlling symptoms. Pharmacological therapy includes drugs that reduce the body's immune response, such as methotrexate (e.g., Rheumatrex, DAVA); azathioprine (e.g., Azasan, Salix); cyclophosphamide (e.g., Cytoxan, Bristol-Myers Squibb); chlorambucil (Leukeran, GlaxoSmithKline); and cyclosporine (e.g., Neoral, Novartis). Skin rashes are treated with corticosteroid creams, and nonsteroidal anti-inflammatory drugs (NSAIDs) are used to control arthritis and pleurisy. Hydroxychloroquine (e.g., Plaquenil, Sanofi), an antimalarial drug, is also used to treat the dermal and arthritic symptoms of SLE.^{2,5}

In March 2011, the FDA approved belimumab (Benlysta, Human Genome Sciences/GlaxoSmithKline), an innovative therapy for patients with SLE. Benlysta was previously known as Lympho-Stat-B.

PHARMACOLOGY

Mechanism of Action¹¹⁻¹³

Belimumab is the first in a new class of drugs known as B-lymphocyte stimulator (BLyS)-specific inhibitors. Belimumab blocks the binding of soluble BLyS, a B-cell survival factor, to receptors on B cells. BLyS is a member of the tumor necrosis factor (TNF) ligand superfamily and contributes to B-cell proliferation and differentiation. Although belimumab does not directly bind to B cells, it inhibits the survival of B cells, including autoreactive B cells, by binding BLyS. It also reduces the differentiation of B cells into immunoglobulin-producing plasma cells.

Pharmacokinetics¹²

The pharmacological profile of belimumab is based on findings from 563 patients who were treated with intravenous (IV) belimumab 10 mg/kg in clinical trials (Table 1). The first three doses were infused at 2-week intervals; infusions were administered at 4-week intervals thereafter.

Belimumab reached a peak concentration (C_{max}) of 313 mcg/mL, and the area-under-the-curve (AUC_{0-∞}) concentration was 3,083 days • mcg/mL. Distribution and terminal half-lives were 1.75 days and 19.4 days, respectively. The systemic clearance was 215 mL/day, and the volume of distribution was 5.29 L.

Pharmacokinetic parameters were not significantly affected by age; most patients (70%) were between 18 and 45 years of age. Belimumab has not been studied in pediatric patients, and limited data exist for elderly patients. Sex and race did not significantly influence pharmacokinetics. The study population was predominantly female (94%) and Caucasian (52%).

The effects of renal impairment on the pharmacokinetics of belimumab were not evaluated; belimumab has been studied in only a limited number of SLE patients with renal impairment. Dosage adjustments in patients with renal impairment are not recommended.

Although elevated creatinine clearance (CrCl) values and the presence of proteinuria (more than 2 g/day) increased the clearance of belimumab, these effects were within the expected range of variability. No formal studies were conducted to examine the effects of hepatic impairment on the drug's pharmacokinetics, and belimumab has not been studied in patients with severe hepatic impairment. Baseline levels of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) did not significantly affect the drug's pharmacokinetics.

Pharmacodynamics¹²

Treatment with belimumab in two clinical trials for 52 weeks significantly reduced circulating CD19+, CD20+, naive, and activated B cells; plasmacytoid cells; and the SLE B-cell subset. Reductions in naive B cells and in the SLE B-cell subset

were also observed at week 8. Although memory cells increased initially, their numbers slowly declined toward the baseline level by week 52.

The clinical relevance of these effects on B cells is unknown. As early as week 8, belimumab therapy led to reductions in immunoglobulin G (IgG) and in anti-dsDNA and to increases in complement (C3 and C4); these effects were sustained through week 52. The clinical relevance of these changes has not been established.

PIVOTAL PHASE 3 STUDIES¹⁴⁻¹⁸

Two pivotal phase 3, multicenter, randomized, double-blind trials—Belimumab in Subjects With Systemic Lupus Erythematosus (BLISS-52 and BLISS-76)—evaluated the efficacy and safety of belimumab plus standard of care in adults with active autoantibody-positive SLE. BLISS-52 included patients in Eastern Europe, Latin America, and the Asia-Pacific region. The BLISS-76 study included patients in North America, Western Europe, and Latin America. The same inclusion and exclusion criteria, treatment regimens, and endpoints were used in both trials.

SLE patients were enrolled in the studies if they had SELENA-SLEDAI scores of 6 or higher, were seropositive for ANAs and/or anti-dsDNA at two independent time points, and had received standard care for at least 30 days. These entry criteria were used to identify active SLE disease and to focus on patients who were more likely to have B-cell dysfunction and to produce autoantibodies.

Table 1 Pharmacokinetic Parameters in Patients With Systemic Lupus Erythematosus After Intravenous Belimumab 10 mg/kg*

Parameter	Population Estimate (N = 563)
Peak concentration (C _{max} , mcg/mL)	313
Area under the curve (AUC _{0-∞} , day • mcg/mL)	3,083
Distribution half-life (days)	1.75
Terminal half-life (days)	19.4
Systemic clearance (mL/day)	215
Volume of distribution (L)	5.29

* Intravenous infusions were administered at 2-week intervals for the first three doses and at 4-week intervals thereafter. (From Benlysta prescribing information.¹²)

Patients with severe lupus nephritis, central nervous system (CNS) lupus, or CNS vasculitis requiring medical treatment within 60 days of study initiation were excluded from the trial. Patients could not use other investigational agents, biologics, anti-tumor necrosis factor (TNF) therapy, IV immunoglobulin, or IV cyclophosphamide.

Baseline demographic characteristics were similar between the two trials, except for patients' scores from the mean Systemic Lupus International Collaborative Clinics/American College of Rheumatology (SLICC/ACR) Damage Index (0.56 in BLISS-52 and 1.00 in BLISS-76) and their scores from baseline treatment. Most patients in both studies were receiving concomitant corticosteroid therapy or two or more classes of SLE drugs, or both.^{14-16,18}

In BLISS-52 and BLISS-76, patients received belimumab 1 mg/kg or 10 mg/kg for 52 weeks or placebo for 76 weeks. Previous treatment (i.e., standard of care) was continued throughout the duration of the studies and could consist of prednisone, antimalarials, NSAIDs, or immunosuppressive agents. Patients were permitted to add or modify antimalarial drugs (up to week 16), immunosuppressive agents (up to week 16), and corticosteroids (up to week 24). Any increase in medication use beyond the allotted dates was considered a treatment failure.^{17,18}

In both trials, the primary efficacy endpoint was the proportion of patients with improvement in disease activity at week 52, compared with baseline, as defined by the SLE Responder Index (SRI). Measures of improvement included:

- an increase of 4 points or more in SELENA-SLEDAI scores.
- no new BILAG A organ domain scores.
- no more than one new BILAG B organ domain score.
- no worsening in Physician's Global Assessment (PGA) scores.

Efficacy Results¹²

In BLISS-52, response rates were analyzed at week 52 in 865 patients. Significantly more patients receiving belimumab 10 mg/kg plus standard of care achieved reductions in disease activity compared with placebo patients (58% vs.

44%, respectively; $P < 0.001$). Moreover, significantly more patients receiving belimumab 1 mg/kg showed significant improvements in disease activity compared with the placebo arm (51% vs. 44%, respectively; $P = 0.013$). At week 24, both groups of patients receiving belimumab 1 mg/kg and 10 mg/kg achieved significant reductions in PGA scores compared with placebo patients (-7.07 and -14.3, respectively; $P \leq 0.05$).

In BLISS-76, 819 SLE patients were evaluated at week 52. Significantly more patients receiving belimumab 10 mg/kg plus standard of care experienced reduced disease activity compared with placebo patients (43% vs. 34%, respectively; $P = 0.021$). However, the belimumab 1-mg/kg arm did not achieve a significant improvement in disease activity compared with the placebo group.

In a secondary assessment, response rates with belimumab were greater than placebo responses at week 76, but the difference was not statistically significant. Patients who received belimumab 10 mg/kg experienced a reduction in disease flares compared with patients who were given placebo; however, this reduction was not considered to be clinically relevant.

Safety Results¹²

In most cases, no dose-related increases in adverse events (AEs) were reported in the clinical trials; therefore, much of the data regarding AEs has been pooled and reported as such.

Infections were the most common AEs associated with belimumab in BLISS-52 and BLISS-76. Infections were reported in 4.8% and 6.3% of patients receiving belimumab 10 mg/kg or placebo, respectively (in BLISS-52) and in 8.1% and 3.6% of patients receiving 10 mg/kg or placebo, respectively (in BLISS-76). Upper respiratory tract infections were the most common type, occurring in more than 5% of belimumab patients.

Infusion reactions occurred in 15.1% of patients receiving belimumab 10 mg/kg, in 15.9% of patients receiving belimumab 1 mg/kg, and in 13.5% of patients receiving placebo.

In BLISS-52, psychiatric AEs, such as depression, insomnia, and anxiety, were reported in 13.8% of patients receiving the 10-mg/kg dose, in 8.7% of those receiving 1 mg/kg, and in 9.4% of those

receiving placebo. In BLISS-76, psychiatric AEs occurred in 16.5%, 19.6%, and 12.0% of patients receiving belimumab 10 mg/kg, 1 mg/kg, or placebo, respectively.

Twelve patients died during the two trials—four who received belimumab 1 mg/kg, five patients who received 10 mg/kg, and three who received placebo. The deaths were associated with infection, lupus flare, cardiovascular disease, malignancy, and suicide.

INDICATIONS AND USAGE¹²

Belimumab is indicated for the treatment of adults with active, autoantibody-positive SLE who are receiving standard therapy. The drug's efficacy has not been evaluated in patients with severe active lupus nephritis or severe active CNS lupus, and it has not been studied in combination with other biologics or with IV cyclophosphamide. Belimumab is not recommended in these situations.

ADVERSE DRUG REACTIONS¹²

Most AEs in the trials were not related to increased doses. The most commonly reported AEs for the recommended dose of 10 mg/kg were nausea, diarrhea, pyrexia, nasopharyngitis, bronchitis, insomnia, extremity pain, depression, migraine, and pharyngitis. In the trials, serious infections (the most serious AEs) occurred in 6% of all belimumab patients and in 5.2% of placebo patients. Rates of treatment discontinuation owing to any AE were 6.2% for the belimumab groups and 7.1% for the placebo groups. Infusion reactions were the most common reason for discontinuing therapy in the belimumab groups (1.6%). Lupus nephritis was the most common cause of stopping therapy in the placebo groups.

In two double-blind, placebo-controlled studies,^{12,18} anti-belimumab antibodies were detected in four of 563 patients (0.7%) who received belimumab 10 mg/kg and in 27 of 559 patients (4.8%) who were treated with belimumab 1 mg/kg. Three patients with anti-belimumab antibodies reported mild infusion reactions, including nausea, erythematous rash, pruritus, eyelid edema, headache, and dyspnea. The observed incidence of antibody positivity depended on factors such as assay sensitivity and specificity, concomitant medication use, and underlying disease. The clinical relevance of the

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presence of anti-belimumab antibodies has not been established.

DRUG INTERACTIONS¹²

No clinically significant drug interactions have been identified during the use of belimumab. In clinical trials, belimumab was given concomitantly with corticosteroids, antimalarial drugs, immunomodulatory and immunosuppressive agents, angiotensin-converting enzyme (ACE) inhibitors, statins, and NSAIDs. None of these agents had clinically significant effects on the pharmacokinetics of belimumab.

The potential effects of belimumab on the pharmacokinetics of other drugs have not been studied. In addition, the administration of belimumab with biologic therapies, including B-cell targeted drugs, or with IV cyclophosphamide has not been studied and is therefore not recommended.

CONTRAINDICATIONS¹²

The use of belimumab is contraindicated in patients who have experienced an anaphylactic reaction to the drug.

WARNINGS AND PRECAUTIONS^{12,18}

In three clinical trials, 14 patients among 2,133 died during the placebo-controlled, double-blind treatment periods—6/674 (0.9%) of the belimumab 10-mg/kg arm, 5/673 (0.7%) of the belimumab 1-mg/kg arm, and 3/675 (0.4%) of the placebo arm. The causes of death included infection, cardiovascular disease, and suicide.

The overall incidence of infection was 71% in treated patients and 67% in the placebo patients. Health care professionals should use caution when administering belimumab to patients with chronic infections. If a new infection develops during belimumab therapy, temporary cessation of treatment is recommended.

Similar to other immunomodulatory therapies, belimumab may increase the risk of malignancy. Hypersensitivity and infusion-site reactions have also been reported. Belimumab should be administered by health care providers who are prepared to manage anaphylactic and infusion reactions. Patients should also receive prophylaxis against infusion and hypersensitivity reactions before treat-

ment begins. Patients should be monitored before and after drug administration.

Psychiatric AEs (depression, insomnia, and anxiety) were reported more frequently with belimumab than with placebo. Two suicides occurred during clinical trials; however, most of these patients with psychiatric AEs had a history of depression or other serious psychiatric disorders.

Belimumab may interfere with the patient's response to immunizations; therefore, live vaccines should not be administered within 30 days before treatment starts or during treatment.

As a Pregnancy Category C medication, belimumab should be prescribed for pregnant women only if the potential benefits outweigh the risks. Women of childbearing age should use adequate contraception for at least 4 months after treatment begins.

Belimumab was excreted in the milk of cynomolgus monkeys. It is not known whether the drug is excreted in human breast milk.

The safety and efficacy of belimumab have not been studied in children. In addition, clinical studies have not included sufficient numbers of patients 65 years of age or older to determine whether their responses differ from those of younger subjects. Therefore, belimumab should be used with caution in elderly patients.

In two clinical trials, response rates for the primary endpoint were lower among African-Americans receiving belimumab, compared with African-Americans receiving placebo. Health care providers should use caution when prescribing belimumab to African-American patients.

DOSAGE AND ADMINISTRATION^{12,18}

Belimumab is intended for IV infusion only. The recommended regimen is 10 mg/kg at 2-week intervals for the first three doses and at 4-week intervals thereafter.

Belimumab is provided as a lyophilized powder in a single-use vial. It should be reconstituted, diluted, and given as an IV infusion over a period of 1 hour. The infusion rate may be slowed or interrupted if the patient experiences an infusion reaction. The infusion must be stopped immediately if a serious hypersensitivity reaction occurs. Belim-

umab should not be infused in the same IV line with other agents.

Although it is recommended that health care professionals premedicate patients against infusion and hypersensitivity reactions before starting treatment, infusion reactions have occurred in similar proportions of patients who received belimumab or placebo in clinical trials. Further, clinical evidence is inadequate for determining whether premedication reduces the incidence or severity of hypersensitivity reactions with belimumab.

COST¹⁹⁻²²

Belimumab is available in 120-mg and 400-mg vials with approximate costs of \$443 and \$1,477, respectively; thus, a weight-based dosing regimen costs approximately \$35,000 per year. Patients who are uninsured or who have other economic barriers to treatment may seek help from the manufacturer's Patient Assistance Program, which provides belimumab at no cost or at a reduced cost to eligible applicants. Insured patients or those who are not eligible for the program but are unable to pay for belimumab may also apply to the manufacturer's Benlysta Gateway program for financial help.

P&T COMMITTEE CONSIDERATIONS

P&T committees, as well as pharmacy benefit management (PBM) organizations, should consider adding belimumab to their formularies for patients with active, autoantibody-positive SLE. In the long term, the drug's cost may be considerably less than the overall costs of SLE and its complications. Belimumab has the potential to reduce disease activity and flares and to decrease the need for other lupus drugs, such as steroids, that have been associated with long-term adverse consequences.

CONCLUSION

SLE is a severely debilitating and potentially fatal disease that can have a significant impact on productivity and quality of life. The novel disease-modifying drug belimumab (Benlysta), when combined with standard of care, has the potential to delay disease progression and improve clinical outcomes.

SLE is also discussed in detail in the *continued on page 226*

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feature article by Dr. Hilas and Dr. Maidhof on page 240.

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