

Rituximab in Rheumatological Diseases: Clinical Efficacy and Safety

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Received: 2026-09-21 · Accepted: 2026-10-17

Abstract

Background: Rituximab depletes CD20-positive B cells and is used in selected autoimmune rheumatic diseases. However, therapeutic benefit differs across diagnoses, and prolonged immune effects require individualized safety assessment. **Objective:** To compare clinical efficacy across rheumatoid arthritis, antineutrophil cytoplasmic antibody-associated vasculitis, and systemic lupus erythematosus, while examining important safety considerations. **Materials and Methods:** A focused narrative synthesis examined five established randomized trials and one pooled long-term safety analysis. Published enrollment, response rates, and infection outcomes were extracted descriptively. No patients were newly recruited, and no pooled treatment effect was calculated because populations, comparators, and endpoints differed substantially. **Results:** In rheumatoid arthritis, rituximab improved responses both after inadequate methotrexate benefit and after tumor necrosis factor inhibitor failure. In the REFLEX trial, 51% of rituximab recipients achieved an American College of Rheumatology 20% response at week 24, compared with 18% receiving placebo. In severe associated vasculitis, complete remission without prednisone at six months occurred in 64% versus 53% with cyclophosphamide-based treatment, meeting noninferiority criteria. Conversely, randomized trials in extrarenal lupus and proliferative lupus nephritis did not demonstrate significant primary-endpoint improvement. Long-term rheumatoid arthritis data reported 3.94 serious infections per 100 patient-years. **Discussion:** These findings support indication-specific prescribing rather than assuming uniform efficacy from B-cell depletion. Infection risk, hepatitis B reactivation, immunoglobulin decline, and impaired vaccine responses remain central considerations. **Conclusion:** Rituximab is an effective option in selected rheumatoid arthritis and vasculitis populations; lupus use requires greater caution when interpreting evidence and balancing alternatives.

Keywords: rituximab, rheumatoid arthritis, vasculitis, systemic lupus erythematosus, B-cell depletion, infection, safety

1. Introduction

B cells contribute to the pathogenesis of several rheumatological diseases through autoantibody production, antigen presentation, cytokine secretion, and interactions with other immune cells. These functions provide a rationale for therapies targeting B-cell populations, particularly when conventional immunosuppressive treatment does not adequately control disease or causes unacceptable toxicity. Rituximab, a chimeric monoclonal antibody directed against CD20, induces depletion of circulating B

cells and has become an important therapeutic option in selected autoimmune rheumatic disorders. Its clinical value, however, varies across diseases, organ manifestations, and treatment settings, requiring an assessment that considers both effectiveness and treatment-related harm.

The strongest evidence supporting rituximab in rheumatology concerns rheumatoid arthritis and antineutrophil cytoplasmic antibody-associated vasculitis. In rheumatoid arthritis, rituximab can improve disease activity and functional outcomes, particularly in seropositive patients with an inadequate response to previous therapies. In granulomatosis with polyangiitis and microscopic polyangiitis, it is an established option for remission induction and maintenance, with treatment selection influenced by disease severity, relapse history, and prior immunosuppressive exposure. Rituximab is also used off-label in selected patients with systemic lupus erythematosus, systemic sclerosis, inflammatory myopathies, and other refractory autoimmune conditions. Evidence in these settings is less uniform, and observational reports of benefit must be interpreted alongside variable or inconclusive randomized trial findings.

Differences in therapeutic response may reflect underlying disease biology as well as clinical heterogeneity. Although CD20-directed treatment depletes many mature B cells, it does not directly eliminate long-lived plasma cells, potentially permitting persistence of pathogenic autoantibodies. Tissue-resident B-cell populations, the kinetics of B-cell repopulation, and concomitant immunosuppression may also influence outcomes. In routine practice, variations in dosing schedules, retreatment criteria, disease duration, and baseline organ damage further complicate comparisons between patients and diagnostic groups. Clinical improvement therefore needs to be evaluated using disease-appropriate measures rather than a single response definition applied indiscriminately across rheumatological conditions.

Safety is a central consideration because patients receiving rituximab frequently have substantial comorbidity and cumulative exposure to glucocorticoids or other immunosuppressants. Infusion-related reactions are recognized adverse events, while infections represent a clinically important concern during follow-up. Repeated treatment may be associated with hypogammaglobulinemia in some patients, and hepatitis B reactivation requires careful pretreatment assessment and appropriate preventive management. Rare complications, including progressive multifocal leukoencephalopathy, warrant awareness without overstating their frequency. Reduced vaccine responsiveness and occasional delayed neutropenia further support individualized monitoring. Distinguishing treatment-associated events from complications of active disease or concomitant therapy remains particularly challenging in observational research.

Despite extensive clinical experience, uncertainties remain regarding the balance of benefit and risk in heterogeneous rheumatology populations, especially among patients underrepresented in controlled trials. Real-world studies can complement trial evidence by examining disease control, glucocorticoid reduction, treatment persistence, and adverse events under routine care conditions. Such evaluations should account for differences in baseline severity and treatment exposure, while avoiding causal interpretations unsupported by their design. The present study aimed to evaluate the clinical effectiveness and safety of rituximab in patients with rheumatological diseases, using disease-specific outcomes where applicable and systematically assessing adverse events during follow-up. A further objective was to explore clinical characteristics associated with response and complications, thereby informing more individualized treatment selection and monitoring.

2. Materials and Methods

This retrospective, observational cohort study was designed to evaluate the clinical effectiveness and safety of rituximab in adults with rheumatoid arthritis, antineutrophil cytoplasmic antibody-associated vasculitis, or systemic lupus erythematosus treated at a tertiary rheumatology service. The proposed study period included treatment initiation between January 2018 and December 2023, with follow-up through December 2024. Institutional ethics approval and a waiver of individual consent for retrospective review were prerequisites for data extraction. Records were to be de-identified before analysis and stored in an access-restricted institutional database.

Eligible patients were aged 18 years or older, had a rheumatologist-confirmed diagnosis supported by the relevant disease classification criteria, and received a first documented rituximab course for active

rheumatological disease during the inclusion period. Patients receiving rituximab primarily for malignancy or another non-rheumatological indication were excluded. Previous rituximab exposure identified in available records also resulted in exclusion. All eligible patients were included without outcome-dependent selection. Follow-up began with the first infusion and continued until death, loss to follow-up, or 12 months, whichever occurred first; absence of a six-month assessment did not exclude patients from safety analyses.

Data were extracted from clinical notes, infusion records, laboratory reports, and hospitalization records using a standardized form. Baseline assessment comprised the closest evaluation within 30 days before treatment. Variables included age, sex, disease duration, organ involvement, smoking status, comorbidities, previous immunosuppressive treatment, concomitant disease-modifying medications, and prednisone-equivalent glucocorticoid dose. Baseline laboratory variables included complete blood count, serum creatinine, liver enzymes, inflammatory markers, and immunoglobulin concentrations. Hepatitis B screening, tuberculosis assessment when clinically indicated, and vaccination documentation were recorded. A second reviewer checked a random 20% of records and all serious adverse events; disagreements were resolved by consensus.

Rituximab dosing was determined by routine clinical care rather than a study-mandated regimen. Treatment generally comprised two 1,000-mg intravenous infusions approximately two weeks apart or four weekly infusions of 375 mg/m², according to indication and clinician judgment. Actual doses, incomplete courses, premedication, and subsequent treatment cycles were documented. Concomitant glucocorticoids, conventional immunosuppressants, antimicrobial prophylaxis, and changes in background treatment were recorded throughout follow-up. Retreatment was treated as a time-varying exposure for descriptive safety summaries rather than evidence of initial treatment failure.

Clinical assessments were evaluated at three, six, and twelve months, using windows of ± 30 , ± 45 , and ± 60 days, respectively. The principal effectiveness endpoint was disease-specific response at six months. For rheumatoid arthritis, response was defined as a moderate or good European Alliance of Associations for Rheumatology response based on the 28-joint Disease Activity Score using erythrocyte sedimentation rate. For vasculitis, remission required a Birmingham Vasculitis Activity Score of zero and prednisone-equivalent dosing of no more than 7.5 mg/day. For systemic lupus erythematosus, response required a decrease of at least four points in the Systemic Lupus Erythematosus Disease Activity Index 2000, without new major-organ involvement or escalation to rescue immunosuppression. These outcomes were reported separately because they represent different clinical constructs. Secondary outcomes included twelve-month response, glucocorticoid reduction, and treatment discontinuation.

Safety outcomes included infusion reactions, serious infections, other serious adverse events, hypogammaglobulinemia, and death. Infusion reactions were events documented during or within 24 hours after infusion. Serious infections required hospitalization, intravenous antimicrobial treatment, or resulted in death. Hypogammaglobulinemia was defined as immunoglobulin G below the laboratory lower reference limit and distinguished as pre-existing or newly detected. Potential hepatitis B reactivation was assessed among patients with available virological monitoring. Event attribution was reviewed clinically, but temporal association alone was not interpreted as causation.

The sample size was determined by the number of eligible records; no confirmatory power calculation was specified. Continuous variables were summarized using means and standard deviations or medians and interquartile ranges, and categorical variables using counts and percentages. Disease-specific response proportions were presented with 95% confidence intervals. Serious infection rates were expressed per 100 patient-years, with follow-up censored at death or last documented contact. Exploratory regression examined associations between response and baseline disease activity, disease duration, glucocorticoid exposure, and previous treatment, only when event counts supported model complexity. Missingness was reported for each outcome; unavailable disease activity scores were not reconstructed from narrative impressions. Complete-case effectiveness analyses were supplemented by a sensitivity analysis classifying missing six-month outcomes as nonresponse. Analyses were exploratory, with emphasis on effect estimates and uncertainty rather than causal treatment comparisons.

3. Results

The following results describe a simulated cohort constructed for manuscript development; they do not represent observed patient data. The cohort comprised 240 adults who received rituximab for refractory or relapsing rheumatological disease. Rheumatoid arthritis accounted for 120 patients (50.0%), antineutrophil cytoplasmic antibody-associated vasculitis for 60 (25.0%), systemic lupus erythematosus for 40 (16.7%), and other systemic autoimmune diseases for 20 (8.3%). The latter group included inflammatory myopathy, systemic sclerosis overlap syndromes, and primary Sjögren syndrome. Median age was 53 years (interquartile range [IQR], 41–64), 168 patients (70.0%) were women, and median disease duration was 7.2 years (IQR, 3.4–12.1). Previous exposure to at least one biological agent was documented in 102 patients (42.5%).

At treatment initiation, 198 patients (82.5%) were receiving oral glucocorticoids and 174 (72.5%) were receiving a concomitant conventional immunosuppressive agent. Among glucocorticoid users, the median prednisone-equivalent dose was 15 mg/day (IQR, 7.5–30). Rituximab was administered as two 1,000-mg infusions separated by 14 days in 192 patients (80.0%) and as four weekly 375-mg/m² infusions in 48 (20.0%). The weekly regimen was used predominantly for vasculitis. Baseline characteristics differed across indications, with older age and more frequent renal impairment in the vasculitis group and a higher proportion of women in the lupus group.

Clinical assessments at month 12 were available for 224 patients (93.3%). Sixteen lacked a month-12 assessment because of death (n = 3), loss to follow-up (n = 8), or transfer of care (n = 5). Using all treated patients as the denominator and classifying missing assessments as nonresponse, 170 patients (70.8%) met their indication-specific response criterion. These comprised moderate or good European Alliance of Associations for Rheumatology responses in rheumatoid arthritis, complete or partial remission in vasculitis, an SLE Responder Index-4 response in lupus, and prespecified disease-specific improvement in other diagnoses. Because these endpoints measure different constructs, the aggregate proportion represents a descriptive summary rather than a uniform efficacy endpoint. Outcomes by indication are presented in Table 1.

Table 1. Simulated clinical outcomes and serious infections through month 12 by treatment indication

Indication	Treated, n	Month-12 assessment, n	Clinical response, n (%)	Patients with serious infection, n (%)
Rheumatoid arthritis	120	112	84 (70.0)	6 (5.0)
ANCA-associated vasculitis	60	56	48 (80.0)	7 (11.7)
Systemic lupus erythematosus	40	38	26 (65.0)	4 (10.0)
Other systemic autoimmune diseases	20	18	12 (60.0)	1 (5.0)
Total	240	224	170 (70.8)	18 (7.5)

All percentages in Table 1 use the treated population within each indication as the denominator. Among patients with an available month-12 assessment, the response proportion was 75.9% (170/224). In rheumatoid arthritis, mean Disease Activity Score in 28 joints using C-reactive protein decreased from 5.7 (standard deviation [SD], 1.0) at baseline to 3.4 (SD, 1.2) among the 112 evaluable patients; their paired mean reduction was 2.3 points (95% confidence interval [CI], 2.1–2.5). Remission was recorded in 32 of 120 treated patients (26.7%). In vasculitis, 42 of 60 patients (70.0%) achieved complete remission, defined as a Birmingham Vasculitis Activity Score of zero, and six achieved partial remission. Among evaluable lupus patients, median SLE Disease Activity Index 2000 decreased from 12 (IQR, 8–18) to 6 (IQR, 2–10).

Glucocorticoid exposure decreased during follow-up. Among 184 baseline glucocorticoid users with paired dose records, the median prednisone-equivalent dose declined from 15 mg/day (IQR, 7.5–30) to 5 mg/day (IQR, 0–10). A reduction of at least 50% was documented in 116 patients (63.0%), and 38

(20.7%) discontinued glucocorticoids. These changes occurred alongside concomitant treatment adjustments and cannot be attributed exclusively to rituximab. Of 176 patients who initially responded by month 6, 28 (15.9%) subsequently experienced a documented flare before month 12; 12 regained response after additional treatment. Repeat rituximab courses were administered to 86 patients (35.8%), including scheduled maintenance in 64 and treatment for recurrent activity in 22.

Total safety follow-up was 223 patient-years. At least one adverse event was recorded in 106 patients (44.2%). Infusion-related reactions occurred in 34 (14.2%), predominantly during the first infusion; most consisted of transient flushing, pruritus, or throat discomfort and resolved with infusion interruption and supportive treatment. Two patients experienced severe reactions requiring permanent discontinuation. Nonserious infections affected 64 patients (26.7%), most commonly involving the upper respiratory or urinary tract. Eighteen patients experienced 22 serious infections, corresponding to 9.9 events per 100 patient-years (95% CI, 6.2–14.9). Pneumonia accounted for ten events, bloodstream infection for four, and other infections for eight.

Among 216 patients with repeat immunoglobulin measurements, 29 (13.4%) developed an IgG concentration below 6 g/L, including eight with concentrations below 4 g/L. Serious infection occurred in six of the 29 patients with low follow-up IgG (20.7%) and 12 of the remaining 211 patients (5.7%); incomplete testing and uncertain temporal relationships limit this comparison. Late-onset neutropenia occurred in five patients (2.1%). Three patients died, two from infection and one from progressive interstitial lung disease. No hepatitis B reactivation or progressive multifocal leukoencephalopathy was documented. Permanent discontinuation attributed to adverse events occurred in 12 patients (5.0%), including eight with serious infection, two with severe infusion reactions, and two with persistent hypogammaglobulinemia and recurrent infections.

4. Discussion

Rituximab occupies an important but disease-dependent position in rheumatological practice, particularly when conventional immunosuppression provides insufficient control or causes unacceptable toxicity. Its clinical value should be assessed through the combined effects on disease activity, organ involvement, glucocorticoid exposure, and treatment-related complications. Improvement in one domain does not necessarily establish an overall benefit, especially in patients with substantial comorbidity or irreversible damage. Accordingly, efficacy and safety require integrated interpretation rather than separate consideration of response rates and adverse events.

The biological rationale for rituximab rests on depletion of CD20-positive B cells, which contribute to autoantibody production, antigen presentation, and inflammatory signalling. Nevertheless, B-cell depletion is not equivalent to elimination of all pathogenic humoral immunity, because long-lived plasma cells generally lack CD20 expression. This distinction helps explain why clinical improvement may occur without normalization of autoantibody concentrations and why serological changes alone are insufficient measures of treatment success. Likewise, persistent symptoms after treatment may reflect structural damage, non-inflammatory pain, or comorbid conditions rather than ongoing immune-mediated disease.

Interpretation of efficacy must account for the heterogeneity of rheumatological indications. Rituximab has established roles in selected patients with rheumatoid arthritis and antineutrophil cytoplasmic antibody-associated vasculitis, whereas its use in other connective tissue diseases is more context-dependent. Combining these conditions within a single analysis can obscure differences in treatment goals, response kinetics, and background therapy. Control of synovitis, remission of vasculitic manifestations, and stabilization of organ-threatening connective tissue disease are not interchangeable outcomes. Disease-specific assessments therefore provide greater clinical meaning than an undifferentiated estimate of overall response.

Glucocorticoid reduction is another important dimension of effectiveness. A sustained decrease in exposure may reduce cumulative metabolic, cardiovascular, skeletal, and infectious harms, even when complete remission is not achieved. However, steroid tapering is influenced by clinician preference, baseline disease severity, and concomitant immunosuppressive treatment. Any apparent steroid-sparing effect should therefore be interpreted alongside objective measures of disease control and the frequency of relapse or rescue therapy. A short observation period may overestimate benefit

if tapering precedes delayed recurrence, while prolonged follow-up is needed to establish whether lower glucocorticoid requirements are maintained across subsequent treatment cycles.

Safety interpretation similarly requires attention to timing and clinical context. Infusion reactions are generally distinguishable from delayed complications and should not be grouped indiscriminately with serious infections. Infection risk may reflect the combined effects of rituximab, preceding immunosuppression, glucocorticoid dose, age, pulmonary disease, and baseline immune status. Repeated treatment can contribute to hypogammaglobulinaemia in susceptible patients, making serial immunoglobulin assessment particularly relevant when infections recur. Pretreatment screening, including evaluation for hepatitis B, and appropriate vaccination planning remain integral to risk mitigation; vaccination responses may be attenuated after B-cell depletion.

These considerations also complicate comparisons between treatment responders and nonresponders. Patients receiving rituximab later in their disease course may have greater treatment resistance, accumulated damage, and prior drug exposure than those treated earlier. Conversely, selective retreatment of patients who initially benefit can make repeated courses appear more effective or better tolerated. Baseline severity, previous biologic therapy, concomitant medications, and follow-up duration should therefore inform interpretation of clinical trajectories. Where observational data are used, adjustment for measured confounders can strengthen inference but cannot eliminate residual confounding or establish that rituximab independently caused improvement.

Safety remains central to interpreting the clinical value of rituximab in rheumatological practice. Disease control must be weighed against the consequences of sustained B-cell depletion, particularly in patients with substantial prior immunosuppressive exposure. Infusion-related reactions are generally manageable with premedication, appropriate infusion rates, and clinical observation, but infectious complications may have greater implications for treatment continuation. Attribution is challenging because infection risk also reflects disease activity, glucocorticoid exposure, comorbidities, and cumulative treatment burden. Consequently, an apparently acceptable safety profile should not be interpreted as evidence of negligible risk.

Risk mitigation should begin before the first treatment course and continue throughout follow-up. Screening for hepatitis B, reviewing vaccination status, and measuring baseline immunoglobulin concentrations are important components of pretreatment assessment. Patients with evidence of previous hepatitis B infection require an individualized prevention strategy, generally including antiviral prophylaxis when receiving B-cell-depleting treatment. Immunoglobulin monitoring becomes particularly relevant with repeated courses or recurrent infections. Vaccination timing also deserves attention because humoral responses may be attenuated after rituximab administration. Decisions regarding antimicrobial prophylaxis should account for the underlying disease, concurrent therapies, and the intensity of immunosuppression rather than rituximab exposure alone.

The potential to reduce glucocorticoid requirements is another clinically meaningful consideration. Sustained disease suppression may permit tapering and thereby lessen treatment-related morbidity, although this benefit cannot be assumed from improvement in disease activity alone. Demonstrating a steroid-sparing effect requires longitudinal dose measurements and consideration of other therapeutic changes. Likewise, symptomatic improvement should be distinguished from prevention of irreversible organ damage. In diseases with renal, pulmonary, or neurological involvement, functional recovery may remain limited despite effective suppression of active inflammation, particularly when treatment begins after structural injury has developed.

Retreatment strategies should balance maintenance of remission against avoidable cumulative exposure. Fixed-interval dosing may be appropriate in selected disease settings, whereas clinically guided retreatment may reduce unnecessary administration in others. Peripheral B-cell measurements and immunoglobulin concentrations can inform assessment, but neither should replace evaluation of disease activity and organ-specific risk. Serological changes may not parallel clinical responses, and residual symptoms may reflect damage, comorbidity, or noninflammatory mechanisms. Persistent symptoms therefore warrant reassessment before additional immunosuppression is prescribed.

Several methodological considerations constrain interpretation of clinical efficacy and safety. Heterogeneity across diagnoses, baseline severity, previous treatment failures, and concomitant medications can obscure disease-specific effects. In nonrandomized comparisons, confounding by indication may disadvantage rituximab because it is often selected for refractory or organ-threatening disease. Conversely, selective continuation among responders can overestimate longer-term benefit. Incomplete follow-up and inconsistent adverse-event documentation may further distort estimates. Rare serious complications require substantially larger populations and longer observation than most individual studies can provide, while absence of an observed event does not establish its absence as a treatment risk.

Future investigations should prioritize disease-specific analyses, standardized outcome definitions, cumulative glucocorticoid exposure, and patient-reported outcomes alongside conventional activity measures. Prospective surveillance should capture recurrent infections, immunoglobulin trajectories, treatment discontinuation, and outcomes after repeated courses. Comparative studies that address baseline differences would help clarify where rituximab offers the greatest net benefit. Overall, rituximab remains an important therapeutic option for selected rheumatological indications, but its value depends on appropriate patient selection, individualized retreatment, and sustained safety monitoring rather than on clinical response alone.

5. Conclusion

Rituximab represents an important therapeutic option for selected patients with rheumatological diseases, particularly when conventional treatment has been insufficient or when disease severity warrants B-cell depletion. Its clinical value should nevertheless be interpreted within the context of the underlying diagnosis, previous treatment exposure, concomitant immunosuppression, and individual susceptibility to adverse events. Efficacy and safety should not be assumed to be uniform across rheumatological conditions or patient subgroups.

The overall therapeutic assessment should extend beyond improvement in disease activity to include sustained disease control, glucocorticoid reduction, preservation of organ function, and patient-reported outcomes. Where these benefits are achieved, rituximab may help reduce the cumulative burden of active disease and prolonged corticosteroid exposure. However, clinical improvement does not necessarily indicate complete suppression of pathogenic immune activity, and relapse remains possible despite an initial response. Retreatment decisions therefore require integration of clinical findings, laboratory assessments, and prior treatment tolerability rather than reliance on a single biomarker or a fixed schedule alone.

Safety remains central to treatment selection and longitudinal management. Infusion reactions, infections, and reductions in immunoglobulin concentrations are clinically relevant concerns, particularly in patients receiving repeated courses or additional immunosuppressive agents. Pretreatment evaluation, including hepatitis B screening, assessment of infection risk, vaccination review, and baseline immunoglobulin measurement, provides an essential foundation for safer use. Continued surveillance should be individualized according to comorbidity, cumulative treatment exposure, and emerging clinical or laboratory abnormalities. Rare but serious complications also require awareness, although their frequency cannot be reliably characterized in studies with limited sample sizes or follow-up.

Interpretation of clinical outcomes must account for potential confounding by indication, differences in background therapy, and heterogeneity in disease manifestations. These factors limit causal attribution and complicate comparisons between diagnostic groups. Larger prospective studies using standardized response definitions, systematic adverse-event ascertainment, and longer observation are needed to clarify optimal retreatment strategies and identify predictors of durable benefit and treatment-related harm.

In conclusion, rituximab can offer meaningful benefit within a carefully selected and closely monitored treatment strategy. Its most appropriate use depends on balancing disease-specific evidence and therapeutic goals against infection risk, immunological vulnerability, and patient preferences through shared clinical decision-making.

AUTHOR INFORMATION

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