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REVIEW ARTICLE

Vitiligo in a rheumatology context: Significance and therapeutic implications

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ABSTRACT

Vitiligo is increasingly recognized as a systemic autoimmune disorder rather than a purely cutaneous disease. Beyond melanocyte destruction and skin depigmentation, accumulating evidence highlights significant genetic, immunologic, and epidemiologic links between vitiligo and systemic autoimmune rheumatic diseases. Shared susceptibility loci, along with convergence on the interferon-gamma (IFN-γ)/Janus kinase–signal transducer and activator of transcription (JAK–STAT) pathway, help explain the increased coexistence of vitiligo with conditions such as rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), Sjögren's syndrome, and systemic sclerosis. Large epidemiologic studies and Mendelian randomization analyses suggest that RA is among the most frequent rheumatic comorbidities in patients with vitiligo. These pathogenic overlaps have direct therapeutic implications. Janus kinase inhibitors, initially developed for inflammatory and hematologic disorders, have demonstrated efficacy in vitiligo and may offer therapeutic benefits in selected patients with concomitant rheumatic disease. However, the strength of evidence varies across agents and indications, ranging from randomized controlled trials to case reports and mechanistic extrapolation. Conversely, paradoxical vitiligo occurring during treatment with biologic disease-modifying antirheumatic drugs (bDMARDs)—particularly tumor necrosis factor-alpha (TNF-α) and interleukin-17 (IL-17) inhibitors—has been increasingly reported, although current evidence remains largely limited to case reports and small observational studies. This focused narrative review explores the immunopathogenic relationship between vitiligo and rheumatic diseases, synthesizes current epidemiologic data, and discusses practical diagnostic and therapeutic considerations relevant to both dermatologists and rheumatologists. It also underscores the importance of multidisciplinary collaboration and individualized therapeutic strategies for patients at the interface between dermatology and rheumatology.

Keywords: Vitiligo; Autoimmune diseases; Rheumatic diseases; JAK–STAT pathway; Multidisciplinary care.

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1. INTRODUCTION

Vitiligo is an autoimmune skin disease characterized by the selective loss of epidermal melanocytes, leading to well-demarcated, depigmented macules and patches (1). With a global prevalence of 0.5–2.0% (2), the condition typically begins in childhood or early adulthood and shows a slight female predominance (2). Its distribution often involves the oral mucosa, distal limbs, and areas subject to friction (3). The two main clinical types are non-segmental (generalized) vitiligo (NSV) and segmental vitiligo (SV), both of which share an immune-mediated aetiology (4). Vitiligo is increasingly understood not as an isolated skin condition, but as a systemic

autoimmune disorder. Its pathogenesis is linked to a combination of genetic predisposition, environmental factors, metabolic stress, and oxidative damage (5). This systemic nature is reflected in its strong associations with other autoimmune diseases. While autoimmune thyroid disease remains the most common comorbidity, occurring in 10–20% of patients (6), the link with rheumatic diseases is particularly robust. Meta-analyses indicate vitiligo patients have approximately 1.8-fold higher odds of RA and 2.0-fold higher odds of SLE (7). Mendelian randomization studies further suggest that vitiligo confers a 47% increased genetic risk for RA and a 22% increased risk for SLE (8). These findings highlight the need for clinical vigilance. The management of vitiligo often requires coordination between dermatology, rheumatology, and endocrinology. This review examines the rheumatologic interface of vitiligo, outlining shared immunopathologic mechanisms, epidemiologic associations, and the resulting cross-disciplinary therapeutic considerations.

2. METHODS

This focused narrative review synthesizes and critically appraises the literature examining the relationship between vitiligo and systemic rheumatic diseases, with particular emphasis on shared immunopathogenic mechanisms and therapeutic implications. A structured search of PubMed/MEDLINE and Scopus was performed for English-language publications up to March 2024 using terms related to vitiligo, autoimmune and rheumatic diseases, epidemiology, pathogenesis, and treatment, including biologic agents and JAK–STAT–targeted therapies. Priority was given to large epidemiologic studies, systematic reviews and meta-analyses, Mendelian randomization analyses, randomized controlled trials, and key mechanistic studies. Case reports and small case series were included selectively to highlight clinically relevant associations or paradoxical treatment effects. The retrieved literature was thematically synthesized, and key findings were summarized narratively and in tables.

This review has limitations inherent to a narrative design. Although the literature search was structured, it was not exhaustive, and the included evidence is heterogeneous in both methodology and quality. Several therapeutic observations, particularly those related to paradoxical vitiligo and systemic JAK inhibitor use, are derived from observational studies, case series, or mechanistic extrapolation rather than high-level comparative evidence. Consequently, conclusions should be interpreted cautiously and viewed primarily as clinically informative rather than definitive.

3. PATHOGENESIS: A SHARED IMMUNOLOGIC FOUNDATION

The pathogenesis of vitiligo is fundamentally autoimmune and is frequently linked to other autoimmune diseases, including rheumatic disorders." (9, 10). This shared susceptibility is rooted in common genetic loci (e.g., PTPN22, NLRP1) and a critical reliance on the interferon-gamma (IFN-γ)/JAK-STAT signalling axis (10). In vitiligo, activated autoreactive cytotoxic $CD8^+$ T cells destroy melanocytes. The production of IFN-γ initiates a JAK-STAT-dependent cascade, leading to the release of chemokines like CXCL9 and CXCL10, which recruit further effector T cells to the skin (11). The efficacy of JAK inhibitors, initially developed for myelofibrosis and subsequently adopted in the management of RA and dermatologic conditions in promoting vitiligo repigmentation provides direct therapeutic evidence of this common mechanism (12).

A key clinical distinction is traditionally drawn between the two main forms of vitiligo. Non-segmental vitiligo (NSV) is widely recognized as a systemic autoimmune disorder, with a well-established association with other autoimmune diseases and shared genetic susceptibility loci (13). Segmental vitiligo (SV), in contrast, typically presents with a unilateral distribution, often stabilizes early, and has historically been considered less frequently associated with systemic autoimmunity (13).

The prevailing pathogenetic model for SV invokes somatic mosaicism, whereby post-zygotic genetic alterations create a localized population of melanocytes with increased immunogenicity, leading to a spatially confined immune response (14). Importantly, this model does not exclude autoimmunity. Histopathological and immunophenotypic studies have demonstrated melanocyte-specific cytotoxic $CD8^+$ T-cell infiltration in active SV lesions, paralleling the immune effector mechanisms observed in NSV and indicating a shared pathway of immune-mediated melanocyte destruction (15).

Consistent with these observations, larger clinical series and case–control studies have shown that although systemic autoimmune comorbidities are significantly less prevalent in SV than in NSV, they are not absent, and mixed phenotypes combining segmental and non-segmental features are increasingly recognized (16–18). Contemporary immunopathogenic models therefore support the concept that SV and NSV differ primarily in their initiating events and anatomical distribution, while converging on common immune effector mechanisms, placing both entities along a spectrum of vitiligo pathogenesis (19–21).

4. ASSOCIATION OF VITILIGO WITH RHEUMATIC DISEASES

Vitiligo frequently coexists with several autoimmune and rheumatic disorders, reflecting common genetic and immunopathogenic pathways (Table1). Large epidemiologic studies confirm that autoimmune thyroid disease is the most prevalent comorbidity, while rheumatoid arthritis (RA), psoriasis/psoriatic arthritis, systemic lupus erythematosus (SLE), Sjögren's syndrome, and systemic sclerosis occur at higher rates than in the general population (3, 22, 23). A recent systematic review and meta-analysis of over 10,000 individuals with vitiligo reported prevalences of autoimmune thyroid disease (14.2%), psoriasis (5.1%), and RA (3.2%), confirming RA as the most frequent rheumatic association after psoriatic disease (22). A large U.S. population-based study similarly identified significant associations between vitiligo and RA, SLE, autoimmune thyroiditis, and systemic sclerosis (23). A nationwide Korean cohort of over 86,000 vitiligo patients documented increased risks of SLE, systemic sclerosis, Sjögren's syndrome, and RA compared to matched controls, although dermatomyositis/polymyositis and ankylosing spondylitis showed no significant association (24). Clinically, these findings highlight the importance of vigilance for systemic symptoms in patients with vitiligo, especially arthralgias, sicca symptoms, photosensitive rashes, and other features suggestive of connective tissue disease.

Table 1. Rheumatic diseases associated with vitiligo.

Rheumatic Disease	Strength of Association	Key Evidence
Rheumatoid arthritis (RA)	Moderate – most common rheumatic comorbidity	Globally increased prevalence (~3.0–3.5%) in vitiligo from multiple meta-analyses(6,16) ; elevated risk consistently shown in U.S. (3,17), Korean(18), and smaller European/Asian cohorts(21).
Psoriasis / Psoriatic arthritis	Moderate	Worldwide meta-analyses show ~5% prevalence(6,16); confirmed across U.S.(3).
Systemic lupus erythematosus (SLE)	Moderate	Increased risk demonstrated in U.S.(3,17) and Korean nationwide data (18), causal link supported by Mendelian randomization (8)
Sjögren's syndrome	Mild–moderate	Higher prevalence reported in Korean population data (18); U.S. data also supports association (3,17) .
Systemic sclerosis	Mild–moderate	Elevated risk in U.S. (3,17) and Korean cohorts (18); sporadic supportive reports from Europe and Middle East.
Dermatomyositis / Polymyositis	Weak	No robust association in large datasets (U.S., Korea); (3,17,18) very limited global evidence.
Ankylosing spondylitis	Weak / Uncertain	Largely nonsignificant association in Korean (18), no genetic correlation per Mendelian randomization (8).

This table presents an original synthesis developed by the authors to summarize epidemiological associations reported in the cited literature. All data are explicitly referenced in parentheses and correspond to the studies listed in the official reference list.

5. CLINICAL SCENARIOS AND MANAGEMENT

The co-occurrence of vitiligo and rheumatic disease presents distinct clinical challenges, often requiring a structured diagnostic and therapeutic approach.

Clinical scenario 1: The vitiligo patient with new musculoskeletal symptoms

A patient with established non-segmental vitiligo (NSV) who presents with new joint pain or swelling warrants a structured rheumatologic evaluation. The history should explore features of systemic autoimmunity, such as prolonged morning stiffness, symmetric small-joint involvement, sicca manifestations, or Raynaud's phenomenon (25). Physical examination should assess for synovitis, inflammatory deformities, and other relevant skin findings (26).

Epidemiologic evidence supports this vigilance. A large population-based cohort study reported a significantly higher incidence of RA among vitiligo patients (adjusted HR ≈ 1.44) (27). However, it is crucial to remember that joint symptoms in a vitiligo patient are not always due to autoimmune arthritis; common non-inflammatory causes like osteoarthritis or fibromyalgia must be considered (Figure1).

Recommended Investigations:

In patients with vitiligo who develop new musculoskeletal symptoms, the initial evaluation should be guided by clinical suspicion of inflammatory disease rather than routine broad screening. Baseline laboratory assessment may include complete blood count (CBC), metabolic panel, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) (28, 29). Serological testing should be selectively

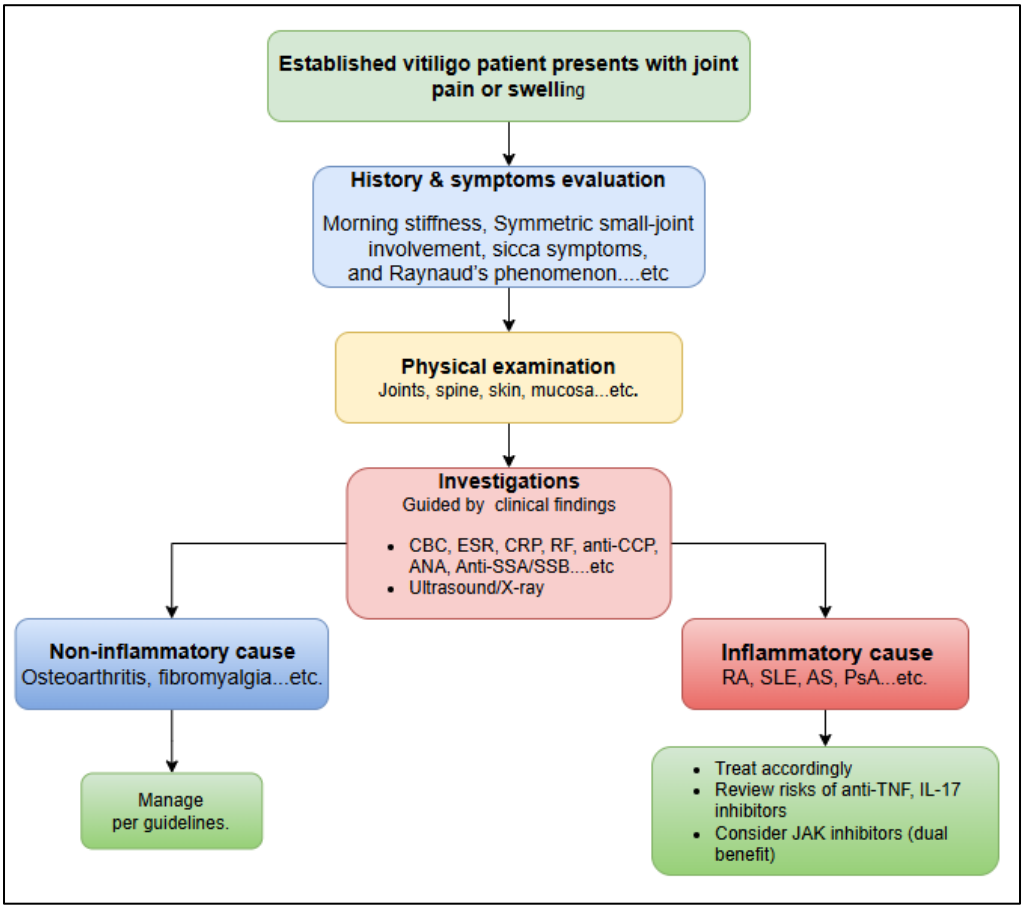

Figure 1. Proposed diagnostic and management algorithm for patients with vitiligo presenting with new-onset musculoskeletal symptoms.

applied according to the suspected underlying condition. Rheumatoid factor (RF) and anti-cyclic citrullinated peptide (anti-CCP) antibodies are appropriate when rheumatoid arthritis is suspected (30), while antinuclear antibody (ANA) testing is indicated when systemic lupus erythematosus or other connective tissue disease is considered (31). In patients with sicca symptoms, anti-SSA/Ro and anti-SSB/La antibodies may be useful (32, 33). Imaging modalities such as musculoskeletal ultrasound or plain radiography may assist in confirming synovitis or structural joint damage when clinically indicated (34, 35). Additional investigations such as urinalysis and complement levels should be reserved for patients with features suggestive of systemic lupus erythematosus (31, 36).

Therapeutic Implications:

When inflammatory arthritis is confirmed in a patient with coexisting vitiligo, treatment selection should follow standard rheumatologic guidelines while accounting for potential dermatologic comorbidity. Because both conditions share involvement of the IFN-γ/JAK-STAT signalling pathway, JAK inhibitors represent a mechanistically relevant therapeutic class; however, the strength of clinical evidence supporting their benefit in vitiligo varies considerably.

Topical and systemic JAK inhibition has shown encouraging results in vitiligo, but the highest level of evidence currently comes from randomized controlled trials of topical ruxolitinib and from baricitinib combined with narrowband ultraviolet B phototherapy (37). In contrast, evidence supporting oral tofacitinib and other systemic JAK inhibitors remains limited mainly to case reports and small uncontrolled series (38, 39). Therefore, JAK inhibitors should be considered when clinically indicated for the underlying rheumatic disease, with potential added dermatologic benefit, rather than as a primary treatment strategy for vitiligo alone.

Conversely, biologic disease-modifying antirheumatic drugs (bDMARDs), particularly TNF-α inhibitors and IL-17 inhibitors, have been associated with vitiligo onset or exacerbation. However, this association is mainly derived from case reports, small series, and pharmacovigilance data (41, 42, 43), and causality remains uncertain. The absolute risk appears low, and these findings should be

interpreted cautiously. Effective biologic therapy should not be discontinued solely on the basis of vitiligo without multidisciplinary evaluation.

Clinical scenario 2:

The rheumatology patient with new-onset vitiligo*. When a patient with established RA or SLE develops new, well-demarcated depigmented patches, the first step is confirming the diagnosis of vitiligo. Under Wood's lamp, vitiligo lesions appear bright, milky white, and sharply circumscribed, helping to distinguish them from mimickers like post-inflammatory hypopigmentation (44, 45).

The clinician must then consider the treatment context, as paradoxical drug-induced vitiligo is a possibility, notably with anti-TNF and anti-IL-17 therapies (46, 47). A dermatology referral is recommended for confirmation and management (Figure 2).

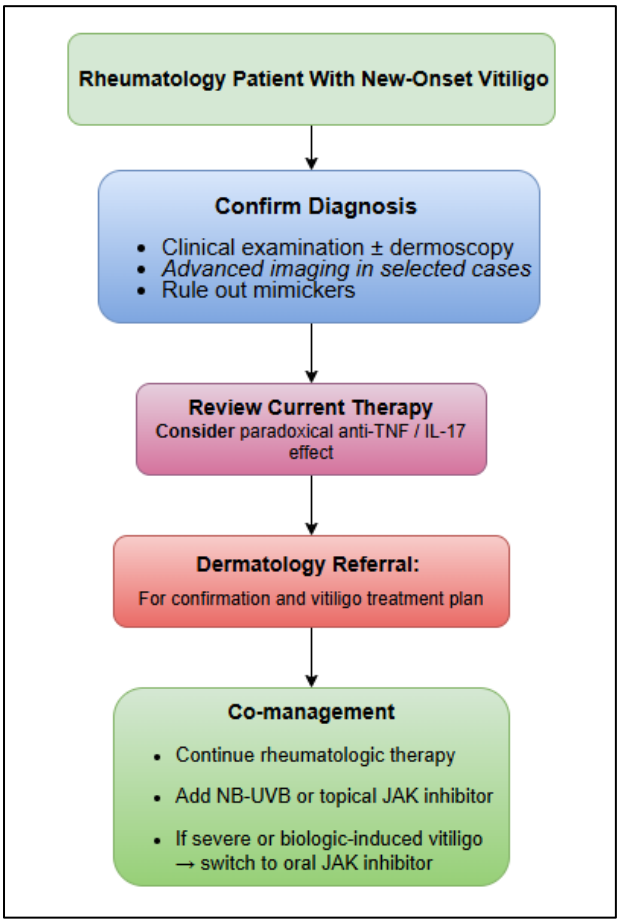

Figure 2. Proposed diagnostic and management algorithm for rheumatology patients presenting with new-onset vitiligo.

Therapeutic Implications:

Rheumatologic therapy should not be stopped solely because of vitiligo. Vitiligo-directed therapy, such as NB-UVB phototherapy or topical JAK inhibitors like ruxolitinib cream (48), can be added while maintaining the rheumatic DMARD. In cases of severe or biologic-induced vitiligo, switching to an oral JAK inhibitor like baricitinib is a reasonable strategy that addresses both conditions (37, 49).

6. THERAPEUTIC INTERSECTIONS AND DILEMMAS

The coexistence of vitiligo and rheumatic diseases presents therapeutic challenges that require balancing efficacy, safety, and differing levels of evidence across drug classes. JAK inhibitors represent a mechanistically attractive therapeutic option due to their inhibition of the IFN-γ/JAK-STAT pathway implicated in both diseases. Nevertheless, the strength of supporting evidence varies. Robust

randomized controlled trial data exist for topical ruxolitinib and for baricitinib combined with narrowband UVB phototherapy, while evidence for other systemic JAK inhibitors remains preliminary and largely observational (37, 48, 51).

Biologic therapies, particularly TNF-α and IL-17 inhibitors, have been associated with paradoxical vitiligo. However, the current evidence base is limited predominantly to case reports, case series, and systematic reviews of such reports (52). As such, these observations should be interpreted as potential signals rather than established causal relationships.

Conventional DMARDs generally have a neutral profile with respect to vitiligo. Methotrexate does not appear to exacerbate depigmentation and may be safely continued when indicated (53). Hydroxychloroquine is also generally considered safe in this context (54). Narrowband UVB phototherapy remains a cornerstone of vitiligo management and can be safely combined with most systemic therapies when clinically appropriate (51) (Table 2).

Table 2. Effects of different drug classes used in rheumatology on vitiligo activity.

Drug Class / Therapy	Effect on Vitiligo	Clinical Implications
JAK Inhibitors (e.g., Baricitinib, Tofacitinib, Ruxolitinib)	**Beneficial**	**Dual-purpose strategy:** Effective for both inflammatory arthritis and vitiligo repigmentation (34). Topical ruxolitinib is approved for vitiligo (42). Oral JAK inhibitors like baricitinib show significant repigmentation in trials (31).
Biologic DMARDs (Anti-TNF, IL-17 Inhibitors)	**Paradoxical Risk** (Induction/Exacerbation)	**Counseling required:** Patients should be informed of the risk of new-onset or worsening vitiligo (36, 37). If it occurs, consider switching to a JAK inhibitor (31, 32); do not stop effective biologic therapy without multidisciplinary discussion.
Conventional DMARDs (Methotrexate, Hydroxychloroquine)	**Neutral**	**Generally safe:** Can be continued. Methotrexate is not superior to NB-UVB alone for vitiligo (47). Hydroxychloroquine does not typically exacerbate vitiligo (48).
NB-UVB Phototherapy	**Beneficial**	**Cornerstone treatment:** A primary therapy for vitiligo that can be safely combined with systemic agents, including JAK inhibitors (31).

This table provides an original synthesis of available evidence derived from clinical trials (31, 42, 47), observational studies (36, 37, 48), and relevant treatment guidelines (34).

7. SCREENING AND COLLABORATIVE CARE

Given the frequent clustering of autoimmunity, dermatologists and rheumatologists must maintain a high level of clinical vigilance.
• *For Dermatologists:* In patients with vitiligo, a focused review for rheumatologic symptoms—joint pain, morning stiffness, sicca features—is recommended. Targeted serologic testing should be guided by clinical suspicion, not performed routinely in asymptomatic individuals. Thyroid function testing (thyroid-stimulating hormone [TSH]) is, however, commonly advised due to the high prevalence of thyroid autoimmunity (55).

• *For Rheumatologists:* All rheumatology patients, especially those on biologic therapies, should undergo basic skin assessment. New depigmented patches should prompt consideration of vitiligo versus other hypo-pigmentary disorders, with dermatology referral as needed (52).

A multidisciplinary approach is paramount. Coordinated care between dermatology and rheumatology improves patient outcomes by ensuring management plans are comprehensive and account for the full spectrum of autoimmune involvement (16).

8. CONCLUSION AND CLINICAL PERSPECTIVES

Vitiligo should no longer be regarded as an isolated cutaneous disorder, but rather as a systemic autoimmune disease with meaningful intersections across rheumatology. Accumulating genetic, immunologic, and epidemiologic evidence demonstrates shared susceptibility loci and convergence on the interferon-γ–driven JAK–STAT signalling pathway, explaining the consistent associations with rheumatic diseases such as rheumatoid arthritis, systemic lupus erythematosus, and other connective tissue disorders. These overlaps are not merely theoretical but translate into tangible clinical and therapeutic consequences.

From a clinical standpoint, the coexistence of vitiligo and rheumatic disease necessitates heightened diagnostic vigilance. Patients with vitiligo presenting with new musculoskeletal symptoms warrant structured rheumatologic evaluation, while individuals with established rheumatic disease, particularly those receiving biologic therapies, should be monitored for the emergence of depigmented

skin lesions. Early recognition facilitates timely diagnosis and avoids misattribution of symptoms to non-immune causes or treatment adverse effects.

Therapeutically, the shared immunopathogenesis creates both opportunities and challenges. JAK inhibitors represent a unique class of agents capable of modulating inflammation in both dermatologic and rheumatologic domains, offering the potential for dual-purpose treatment in selected patients. Conversely, paradoxical induction or exacerbation of vitiligo with certain biologic DMARDs underscores the need for individualized treatment decisions and multidisciplinary discussion rather than reflexive discontinuation of effective rheumatologic therapy.

Limitations

This review has inherent limitations related to its narrative design. Although a structured literature search was performed, it was not exhaustive, and therefore some relevant studies may not have been captured. In addition, the evidence base is heterogeneous, including randomized controlled trials, observational studies, mechanistic research, and case reports. As a result, some therapeutic interpretations, particularly those involving JAK inhibitors and paradoxical vitiligo associated with biologic therapies, should be considered hypothesis-generating rather than definitive conclusions. Ultimately, optimal management of patients at the interface of vitiligo and rheumatology depends on close collaboration between dermatologists and rheumatologists. An integrated, patient-centred approach that acknowledges the systemic nature of vitiligo and its therapeutic intersections with rheumatic disease is essential to improve outcomes and minimize iatrogenic complications. Future research should focus on prospective studies and real-world data to refine screening strategies and clarify the long-term impact of targeted immunomodulatory therapies across this shared autoimmune spectrum.

List of abbreviations

ANA: Antinuclear Antibody
Anti-CCP: Anti-Cyclic Citrullinated Peptide
bDMARD: Biologic Disease-Modifying Antirheumatic Drug
CBC: Complete Blood Count
CRP: C-Reactive Protein
CTD: Connective Tissue Disease
DMARD: Disease-Modifying Antirheumatic Drug
EULAR: European Alliance of Associations for Rheumatology
HR: Hazard Ratio
IFN-γ: Interferon-Gamma
IL-17: Interleukin-17
JAK: Janus Kinase
NB-UVB: Narrowband Ultraviolet B
NSV: Non-Segmental Vitiligo
RA: Rheumatoid Arthritis
RF: Rheumatoid Factor
SLE: Systemic Lupus Erythematosus
STAT: Signal Transducer and Activator of Transcription
SV: Segmental Vitiligo
TNF-α: Tumor Necrosis Factor-Alpha
TSH: Thyroid-Stimulating Hormone
VASI: Vitiligo Area Scoring Index

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