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Narrative Review

Integrated Mechanisms and Clinical Challenges of Rheumatic Diseases in Older Adults: A Narrative Review

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Abstract

Objectives: To investigate rheumatologic diseases in older adults and examine how age-related physiological changes influence disease presentation, progression, and treatment response.

Design: Narrative review.

Setting(s): Global.

Outcome Measures: Current evidence from immunology, epidemiology, gerontology, and clinical medicine to present a comprehensive overview of rheumatologic diseases in older adults.

Results: Aging-related changes, such as immunosenescence, sarcopenia, connective tissue degeneration, and metabolic alterations, contributed to increased prevalence, atypical presentations, and complex management of rheumatic diseases in older adults. Moreover, therapeutic decisions were influenced by multimorbidity, frailty, polypharmacy, and altered pharmacokinetics. Additionally, individualized pharmacologic therapy, non-pharmacologic interventions, and multidisciplinary care were found to be essential. Emerging directions included biomarker discovery, personalized medicine, and telemedicine integration.

Conclusions: Overall, rheumatologic diseases in older adults require individualized, multidisciplinary management strategies that take into account for age-associated physiological and clinical complexities. Furthermore, advances in personalized approaches, geriatric integration, and research into biomarkers and treatment optimization have the potential to improve outcomes and quality of life in this growing patient population. Continued research and tailored clinical frameworks are also imperative for addressing the unique challenges of geriatric rheumatology.

Keywords: Geriatric rheumatology, Immunosenescence, Inflammaging, Aging

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Introduction

The global demographic shift toward an aging population has serious implications for healthcare systems, particularly in relation to chronic non-communicable diseases that disproportionately affect older adults. Specifically, rheumatologic diseases represent a leading cause of disability in individuals aged 60 years and above, substantially contributing to pain, functional decline, healthcare utilization, and diminished quality of life.[1] The interplay of aging-related biological processes, including immunosenescence, chronic low-grade inflammation (inflammaging), sarcopenia, and altered bone and cartilage homeostasis, creates a unique physiological environment that predisposes older adults to rheumatic disorders and modifies their clinical course (Figure 1).[2] In addition, atypical presentations, overlapping conditions, and comorbidities frequently complicate diagnostic evaluation. Furthermore, multimorbidity, frailty, and polypharmacy considerably influence therapeutic strategies, as standard treatment paradigms may not fully apply to elderly patients due to altered pharmacokinetics, heightened susceptibility to adverse events, and challenges related to long-term adherence.[3] Despite the substantial burden of rheumatic disease in this population, geriatric rheumatology remains underrepresented in research, and various clinical trials exclude individuals over 75 years of age.

Given the above-mentioned discussions, this narrative review seeks to synthesize current evidence obtained from epidemiology, immunology, clinical medicine, and gerontology in order to provide a thorough overview of rheumatologic diseases in older adults. Moreover,

the objective is to offer clinicians, researchers, and healthcare policymakers a cohesive summary of the prevalence, mechanisms, diagnosis, and management of these conditions while highlighting the challenges and opportunities unique to geriatric populations.

Methods

In this narrative review, a literature search was systematically conducted to identify relevant publications on rheumatologic diseases in older adults. The search was performed in major scientific databases, including PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar. Several keywords and combinations were used, including "geriatric rheumatology," "aging," "immunosenescence," "inflammaging," "rheumatic diseases in older adults," "elderly rheumatoid arthritis," "late-onset autoimmune diseases," "vasculitis in the elderly," "osteoarthritis aging," and condition-specific terms (e.g., "elderly gout," "CPPD aging," "polymyalgia rheumatica," and "giant cell arteritis"). In addition, the search covered studies published between 2000 and 2024, with additional seminal articles included, irrespective of date, when essential for the conceptual background. Furthermore, reference lists of key articles were screened to identify further relevant sources. It is noteworthy that priority was given to peer-reviewed original studies, systematic reviews, meta-analyses, and major clinical guidelines.

Physiological and Immunological Changes in Aging Relevant to Rheumatologic Diseases

Aging is associated with extensive alterations in immune function, collectively termed immunosenescence, which encompasses functional declines in both innate and adaptive immunity. These changes include reduced T-cell receptor diversity, diminished B-cell production, impaired antigen presentation, decreased neutrophil function, and dysregulated cytokine signaling. [4, 5] One of the notable consequences of immunosenescence is the emergence of a chronic, low-grade inflammatory state, which is referred to as inflammaging and is characterized by elevated circulating levels of interleukin-6, tumor necrosis factor-alpha, and C-reactive protein. [2] This inflammatory milieu contributes to the pathogenesis and progression of various rheumatologic diseases, including osteoarthritis (OA), gout, rheumatoid arthritis (RA), and vasculitides (Table 1).

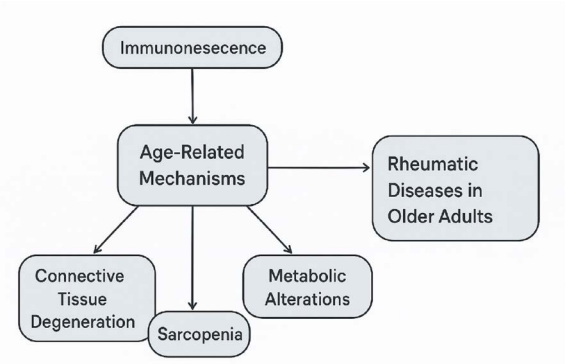

Figure 1. A Conceptual Framework About Rheumatologic Diseases in Older Adults

Beyond immunological changes, aging noticeably affects connective tissue structure and function. Cartilage gradually becomes thinner and less resilient due to reduced proteoglycan content, while subchondral bone undergoes sclerosis and microstructural deterioration.[6,7] In addition, tendons and ligaments lose elasticity, and skeletal muscle mass declines, resulting in sarcopenia, which exacerbates joint instability and functional impairment.[8,9] These changes not only predispose individuals to degenerative diseases (e.g., OA) but also modify the presentation of inflammatory disorders, thereby complicating diagnosis.

Metabolic and endocrine alterations, including reduced renal function, altered uric acid clearance, vitamin D deficiency, and hormonal shifts, further influence susceptibility to rheumatic diseases. Further, age-related decline in renal urate excretion plays a key role in gout pathogenesis, [10, 11] whereas impaired calcium regulation predisposes older adults to calcium pyrophosphate deposition disease (CPPD).[12,13] Collectively, these physiological changes create a landscape in which rheumatologic conditions are both more prevalent and more complex to manage.

Major Rheumatologic Diseases in the Elderly

Osteoarthritis. OA is the most prevalent rheumatologic disorder in older adults, affecting over 70% of individuals aged 65 and above and serving as a major cause of disability worldwide.[6,14] The pathogenesis of OA involves an intricate interplay of mechanical loading, age-related cartilage degeneration, metabolic alterations, subchondral bone remodeling, and synovial inflammation. With aging, chondrocytes demonstrate impaired regenerative

Table 1. Pathophysiology of Aging Relevant to Rheumatology

Mechanism	Key Features	Impact on Disease
Immunosenescence	Decreased T/B cell function and decreased antigen presentation	Impaired immune response and delayed diagnosis
Inflammaging	Increased IL-6, TNF-α, and CRP	Acceleration of OA, RA, and GCA through chronic low-grade inflammation
Sarcopenia	Decreased muscle mass and strength	Joint instability and worsened disability
Connective tissue changes	Decreased cartilage hydration and decreased tendon elasticity	OA progression and altered presentation of inflammatory diseases
Metabolic/endocrine	Decreased renal urate excretion and vitamin D deficiency	Gout and CPPD susceptibility

Note. TNF-α: Tumor necrosis factor-alpha; IL: Interleukin; CRP: C-reactive protein; OA: *Osteoarthritis*; RA: Rheumatoid arthritis; GCA: Giant cell arteritis; CPPD: Calcium pyrophosphate deposition disease.

capacity and increased vulnerability to oxidative stress,[7,15] leading to progressive joint-space narrowing, osteophyte formation, and low-grade synovitis.

Clinically, OA presents with chronic joint pain, stiffness, crepitus, and functional limitation, most commonly influencing weight-bearing joints. Diagnosis relies on clinical evaluation supplemented by radiographic findings.

Management in older adults requires an individualized and multimodal approach. Core non-pharmacologic strategies include weight reduction, structured exercise programs, physiotherapy, and assistive devices.[14,16] Moreover, pharmacologic options encompass acetaminophen, topical nonsteroidal anti-inflammatory drugs (NSAIDs), oral NSAIDs (with caution due to cardiovascular and gastrointestinal risks), and intra-articular corticosteroids. Opioids are generally discouraged because of their potential to cause cognitive impairments, falls, and dependence. Although joint replacement surgery remains an effective intervention for advanced disease, it involves increased perioperative risks in frail or comorbid individuals.

Rheumatoid Arthritis. RA in older adults presents in two forms: elderly-onset RA, which begins after age 60, and long-standing RA persisting into older adulthood. Elderly-onset RA represents 10–33% of RA cases and consistently exhibits distinct characteristics, including acute onset, predominant involvement of large joints and the shoulder girdle, systemic inflammation, and a higher prevalence of seronegative disease.[17–19] These features emerge from the combined effects of immunosenescence, inflammaging, and altered cytokine profiles.

Older adults with RA frequently experience accelerated functional declines due to coexisting OA, sarcopenia, and degenerative spine disease.[9] Furthermore, diagnosis may be delayed due to a lower prevalence of traditional biomarkers (e.g., rheumatoid factor and anti-cyclic citrullinated peptide) in late-onset disease.[19] Additionally, symptom overlap with polymyalgia rheumatica (PMR) or degenerative disorders complicates assessment.

Management parallels that of younger adults but requires careful adjustment. Disease-modifying antirheumatic drugs (i.e., methotrexate, hydroxychloroquine, and sulfasalazine) remain first-line therapies. Likewise, biologic agents and Janus kinase inhibitors are reserved for refractory disease.[17] Given the heightened risk of infections, cytopenias, and organ toxicities, older adults benefit from conservative dosing and close monitoring ("start low and go slow").[3,20] Comorbidities such as renal insufficiency, cardiovascular disease, and pulmonary pathology further constrain therapeutic choices. Although glucocorticoids may be used for rapid symptom control, they should be minimized.

Polymyalgia Rheumatica. PMR is among the most common inflammatory rheumatic disorders in individuals over 50, with peak incidence between ages 70 and 80.[21,22] PMR is characterized by severe pain and stiffness in the shoulder and pelvic girdles, especially in the morning. The pathogenesis of this disorder is attributed to immune dysregulation, genetic predisposition, and heightened interleukin-6–mediated inflammation.[23]

In addition, its diagnosis is clinical, supported by elevated erythrocyte sedimentation rate and C-reactive protein, although normal values do not exclude the disease. Ultrasound may reveal subdeltoid bursitis or synovitis.

Low-to-moderate doses of glucocorticoids constitute first-line therapy, with rapid clinical improvements often providing diagnostic confirmation.[21] Considering that older adults are at increased risk of steroid-related adverse effects, tapering should occur as soon as feasible. Methotrexate may also reduce relapse risk in patients requiring prolonged steroid therapy. [23] Moreover, regular follow-up is essential due to the association with giant cell arteritis (GCA).

Giant Cell Arteritis. GCA is a granulomatous vasculitis that primarily affects individuals aged 50 and older, with incidence rising sharply after age 65.[24–26] Its pathogenesis involves the immune-mediated inflammation of medium and large arteries, driven by dendritic cell activation, T-cell responses, and vascular remodeling.[27]

Additionally, the clinical manifestations of GCA include new-onset headache, jaw claudication, scalp tenderness, visual symptoms, and constitutional features. Untreated GCA is considered a medical emergency because it may cause irreversible blindness.

Diagnosis relies on clinical findings and elevated inflammatory markers, supplemented by imaging modalities such as ultrasound, magnetic resonance imaging, or positron emission tomography–computed tomography. Temporal artery biopsy remains the gold standard. Furthermore, immediate initiation of high-dose glucocorticoids is critical. [26] Similarly, tocilizumab has emerged as an effective steroid-sparing therapy, particularly valuable in elderly patients at high risk of glucocorticoid toxicity.

Gout. It is highly prevalent in older adults due to age-related reductions in renal urate excretion, comorbidities (e.g., hypertension and chronic kidney disease), and frequent use of diuretics and urate-raising medications.[10,28,29] The disease results from monosodium urate crystal deposition in joints, which activates the nucleotide-binding domain and leucine-rich repeat protein 3 inflammasome and induces acute inflammation. In older adults, gout frequently presents with polyarticular involvement and atypical sites, including the hands.[30]

The diagnosis of gout is dependent on synovial fluid analysis, demonstrating negatively birefringent urate crystals, although this may not always be feasible. Serum uric acid levels may be normal during flares.

Its management includes NSAIDs, colchicine, or corticosteroids, with NSAIDs used cautiously due to renal, gastrointestinal, and cardiovascular risks. Further, long-term urate-lowering therapy with allopurinol or febuxostat is indicated in recurrent gout, with dose

adjustment based on renal function. Moreover, lifestyle modification and hydration support long-term control.

Calcium Pyrophosphate Deposition Disease. CPPD is strongly age-associated and represents one of the most common crystal arthropathies in older adults.[12, 13] The disease arises from the deposition of calcium pyrophosphate crystals in joint tissues, leading to acute pseudogout, chronic arthropathy, or asymptomatic chondrocalcinosis. Additionally, age-related chondrocyte dysfunction and impaired pyrophosphate metabolism contribute to disease development.

Clinical presentations overlap with OA and RA. The diagnosis of this disease depends on synovial fluid analysis showing positively birefringent crystals or imaging evidence of chondrocalcinosis.

In addition, CPPD management encompasses NSAIDs, colchicine, and corticosteroids for acute attacks. Furthermore, chronic disease may require prophylactic colchicine and treatment of metabolic contributors.

Systemic Lupus Erythematosus. Systemic lupus erythematosus with onset after age 50 accounts for 12–20% of cases and presents with a more insidious onset and a higher prevalence of constitutional symptoms, arthritis, and serositis, but less frequent cutaneous and renal involvement.[31–35] Immunosenescence contributes to altered B-cell and T-cell responses and lower autoantibody titers, which may delay diagnosis.[35]

Hydroxychloroquine remains foundational therapy for this disease, while glucocorticoids and immunosuppressants are used for moderate to severe cases. Moreover, older adults require vigilant monitoring for drug toxicity, infection, osteoporosis, and cardiovascular complications.[20]

Sjögren's Syndrome. Sjögren's syndrome in the elderly commonly presents with sicca symptoms, fatigue, arthralgia, and neuropathy. In addition, age-related dryness and comorbidities may obscure diagnosis, and anti-Ro/SSA antibodies are less frequently detected.[36,37] Further, lymphoma risk may be elevated.

Management of this syndrome encompasses symptomatic treatments and immunosuppressive therapy for systemic involvement, with attention to infection risk, dental health, and malignancy screening.

Systemic Sclerosis. Systemic sclerosis in older adults often demonstrates limited cutaneous disease but greater vascular and pulmonary complications.[38,39] Age-related endothelial dysfunction and fibrosis have a role in disease severity.

Its management requires vasodilators for Raynaud phenomenon, proton pump inhibitors for reflux, and immunosuppressants for interstitial lung disease, with careful consideration of the risks of immunosuppression in older patients.[40,41]

Idiopathic Inflammatory Myopathies. Idiopathic inflammatory myopathies, including polymyositis, dermatomyositis, and inclusion body myositis, occur in elderly individuals and often present with an insidious onset. Inclusion body myositis, which is common in older men, responds poorly to immunosuppression.[42]

Glucocorticoids, immunosuppressive therapy, physical rehabilitation, fall prevention, and nutritional assessment are necessary for controlling this group of disorders.

Vasculitides in the Elderly. Beyond GCA, anti-neutrophil cytoplasmic antibody-associated vasculitides occur more frequently in older adults and repeatedly present with renal impairments, pulmonary involvement, and neuropathy.[43–47] The diagnosis of vasculitides requires high suspicion due to symptom overlap with infection and malignancy.

Moreover, their management involves glucocorticoids and immunosuppressants, such as rituximab or cyclophosphamide, adjusted for increased toxicity risk.

Diagnostic Challenges in Elderly Rheumatology Patients

The diagnosis of rheumatologic diseases in older adults is complicated by overlapping symptoms with common geriatric syndromes. Similarly, laboratory markers may be unreliable due to immunosenescence, and multimorbidity or medication side effects may mimic rheumatic disease. Degenerative changes can confound imaging findings, while cognitive or sensory impairments may limit history-taking. A holistic, multidisciplinary approach is often required for diagnosis.

Principles of Management in Elderly Patients With Rheumatologic Conditions

Therapeutic strategies must account for physiological changes, comorbidities, functional status, and polypharmacy. Core principles are summarized as follows (Figure 2):

- Initiating medications at reduced doses with gradual titration
- Minimizing glucocorticoid exposure
- Using NSAIDs cautiously and with gastroprotection
- Integrating non-pharmacologic therapies, including exercise and physical therapy
- Assessing fall risk, nutrition, bone health, and functional capacity
- Engaging multidisciplinary teams (rheumatology, geriatrics, physiotherapy, occupational therapy, and nutrition).

Telemedicine and remote monitoring technologies show promise for improving access to care.

Quality of Life, Disability, and Functional Decline

Rheumatic diseases significantly impair the quality of life through pain, stiffness, fatigue, and loss of mobility. In addition, functional decline accelerates in the presence of frailty or sarcopenia. Psychological factors (e.g., depression, anxiety, and sleep disturbances) often contribute to disability but remain underrecognized. Interventions such as exercise programs, balance training, mental health support, and comprehensive pain management are essential for controlling these diseases.

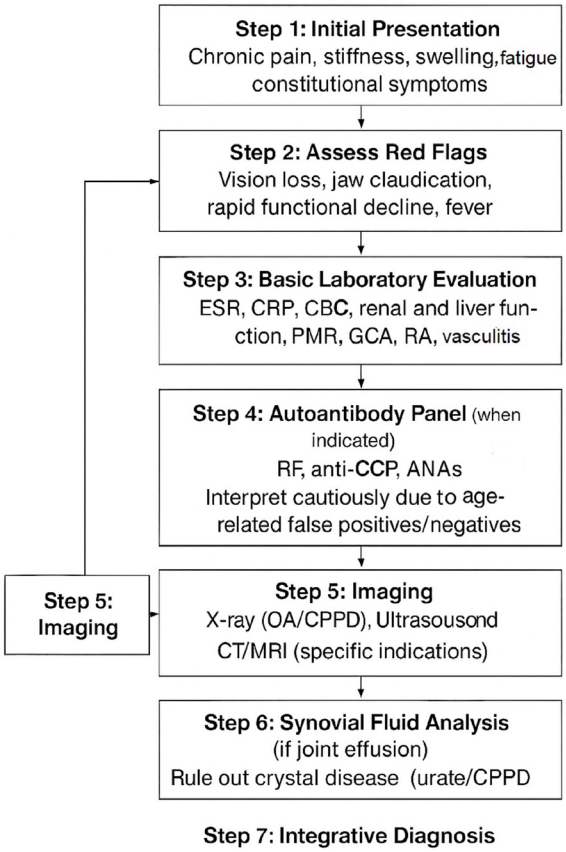

Figure 2. Diagnostic Flowchart for Evaluating Rheumatic Diseases in Older Adults
Note. ESR: Erythrocyte sedimentation rate; CRP: *C-reactive protein*; CBC: Complete blood count; PMR: Polymyalgia rheumatica; GCA: Giant cell arteritis; RA: Rheumatoid arthritis; RF: Rheumatoid factor; CCP: Cyclic citrullinated peptide; ANAs: Antinuclear antibodies; OA: Osteoarthritis; CPPD: Calcium pyrophosphate deposition disease; CT: Computed tomography; MRI: Magnetic resonance imaging

Discussion

This narrative review integrated the major biological, clinical, and epidemiological dimensions of rheumatic diseases in older adults, highlighting the unifying mechanisms that shape their presentation and management. The findings demonstrated that OA, RA, PMR, gout, CPPD, and systemic autoimmune diseases share several age-related drivers, including immunosenescence, inflammaging, connective tissue degeneration, sarcopenia, and metabolic dysregulation. Although these diseases are traditionally discussed in isolation, the evidence indicates that they arise from overlapping physiological pathways that interact with comorbidities, frailty, and polypharmacy to produce complex clinical profiles in geriatric populations.

Among conditions, diagnostic challenges were consistently linked to atypical symptomatology, altered biomarker patterns, and the effects of multimorbidity. These challenges demonstrate the need for integrated assessment strategies that combine clinical evaluation with imaging, laboratory data, and functional status. The diagnostic flowchart presented in this review is expected to provide clinicians with a structured approach to navigating these complexities. Similarly, management principles across diseases converge on cautious pharmacologic decision-making, minimization of glucocorticoid exposure, and incorporation of non-pharmacologic interventions tailored to functional capacity and frailty.

Likewise, the synthesis presented in this study underscores that geriatric rheumatology requires a shift from disease-centered frameworks to mechanism-oriented and patient-centered paradigms. Therefore, future research should prioritize biomarker development, stratified treatment approaches, and clinical trials inclusive of older adults to address the persistent knowledge gaps in this field. Overall, integrating age-related pathophysiology with disease-specific insights offers a more comprehensive foundation for improving diagnosis, treatment, and quality of life in older patients with rheumatic disorders.

Conclusions

In general, rheumatologic diseases are considered a major and growing healthcare challenge in aging societies. The interplay of immunosenescence, inflammaging, multimorbidity, and functional decline produces distinct disease patterns in older adults and necessitates individualized, multidisciplinary care. Advances in personalized medicine, geriatric integration, and biomarker research hold promise for improving outcomes. Overall, this review indicates the need for continued research, enhanced diagnostic strategies, and patient-centered approaches to optimize the management of rheumatic diseases in older populations.

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The authors have nothing to report.

Authors' Contributions

Conceptualization: Sepideh Tahsini Tekantapeh.
Data Curation: Sepideh Tahsini Tekantapeh.
Investigation: Sepideh Tahsini Tekantapeh.
Methodology: Soodabeh Davaran.
Project Administration: Sepideh Tahsini Tekantapeh.
Resources: Sepideh Tahsini Tekantapeh.
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Supervision: Soodabeh Davaran.
Validation: Sepideh Tahsini Tekantapeh.
Visualization: Sepideh Tahsini Tekantapeh.
Writing—Original Draft: Sepideh Tahsini Tekantapeh, Soodabeh Davaran.
Writing—Review & Editing: Sepideh Tahsini Tekantapeh.

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Data Availability Statement

The patient details are available in the electronic medical records and can be made available upon reasonable request from the authors.

Ethical approval

Not applicable.

Consent for publication

Considering that this was a review study, there was no need to obtain informed consent.

Conflict of interests

The authors declare they have no conflict of interests.

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