Clinical Insights | AGING AND HEALTH

Evaluating Inflammatory Joint Pain in Older Adults—Practical Diagnostic Clues for Primary Care Clinicians

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Arthritis is a leading cause of pain and disability that affects nearly one-third of older US adults (age ≥65 years). While osteoarthritis (OA) predominates, many have inflammatory arthritis (IA), including rheumatoid arthritis (RA), spondyloarthritis, and crystal arthropathies.[1] Recognition of IA is frequently delayed for longer than a year in older adults due to atypical and overlapping presentations.[2] Untreated IA can lead to irreversible damage, functional decline, and prolonged glucocorticoid exposure. Joint pain is often initially evaluated by primary care clinicians; therefore, timely recognition and early management are essential. This article highlights unique aspects of IA in older adults and offers practical guidance.

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When to Suspect IA

The initial step in evaluating joint pain is distinguishing inflammatory from noninflammatory patterns. IA pain is accompanied by prolonged morning stiffness (>1 hour), nocturnal pain, and improvement with activity. Pain with passive motion, effusions, and joint warmth also favor inflammation. By contrast, OA pain typically worsens with use and improves with rest. The **Figure** summarizes key features, including temporality and typical joint distribution, highlighting diagnostic clues relevant to older adults. Constitutional symptoms, such as weight loss, fatigue, or low appetite, may present with IA and should not be misattributed to normal aging, contributing to diagnostic delays.[3]

IA Mimics and Diagnostic Pitfalls in Older Adults

Once IA is suspected, clinicians should consider conditions that may present atypically in older adults. RA can present de novo in later life, with incidence peaking in the seventh to eighth decade. In older adults, RA may be seronegative and preferentially involve the shoulders or large joints, producing proximal pain and morning stiffness that can resemble polymyalgia rheumatica (PMR). Although PMR is an important diagnostic consideration in this age group, it does not cause true synovitis. Symmetric synovitis of the hands or wrists, elevated inflammatory markers, and progressive functional impairment support a diagnosis of RA, even when PMR-like proximal stiffness predominates early in the disease course.

Crystal arthropathies are common in older adults, presenting as acute or subacute, asymmetric monoarticular or oligoarticular episodes, and may occur concurrently with other IAs. Gout often affects the feet (podagra), knees, or wrists, while calcium pyrophosphate deposition disease commonly involves the knees and wrists and can mimic RA with polyarticular involvement.

New-onset spondyloarthritis, such as psoriatic arthritis or axial spondyloarthritis, after age 70 years is uncommon, while degenerative spine disease is far more prevalent. In individuals with established spondyloarthritis, sudden worsening back pain should prompt evaluation for fragility fracture. Remitting seronegative symmetrical synovitis with pitting edema (RS3PE) is an older-onset inflammatory syndrome characterized by acute bilateral synovitis of the hands with dorsal pitting edema. Typically, seronegative and steroid-responsive, RS3PE may be associated with underlying cancer. Clinicians should carefully consider occult cancer in older adults with atypical or refractory IA, disproportionate constitutional symptoms, unexplained weight loss, or poor response to therapy.

Diagnostic Approach to Suspected IA

Laboratory evaluation for IA includes inflammatory markers, erythrocyte sedimentation rate, and C-reactive protein levels, which can support clinical suspicion but are nonspecific. The erythrocyte sedimentation rate may be modestly elevated in otherwise healthy older adults (eg, 30-40 mm/h) or in those with anemia; therefore, values must be interpreted considering the clinical context.

Rheumatoid factor and anticyclic citrullinated peptide antibodies are helpful when positive, although seronegative disease is more common in older adults. Antinuclear antibody and human leukocyte antigen–B27 testing should be reserved for individuals with symptoms that suggest connective tissue diseases or spondyloarthritis; however, positive results are rarely diagnostic in isolation.

Plain radiography can identify degenerative changes, erosions, chondrocalcinosis, or other structural causes of pain, but should not be used to exclude IA. Radiographic OA is common in older adults and can coexist with IA. Therefore, when IA is suspected but difficult to assess clinically, musculoskeletal ultrasonography or magnetic resonance imaging should be sought to evaluate for joint inflammation. Joint aspiration is essential when an effusion is present to rapidly clarify diagnosis. With acute monoarticular effusion, aspiration should not be delayed, as septic arthritis may present without high fever or leukocytosis due to blunted inflammatory responses in older adults and can lead rapidly to permanent joint damage.

Management Principles and the Risks of Glucocorticoids

When IA is strongly suspected and access to rheumatology is delayed, initiation of a disease-modifying antirheumatic drug (DMARD) is appropriate and adheres with guidelines.[4] Age should not preclude use of DMARDs, as these therapies are generally safe and effective in older adults. Methotrexate (MTX) remains first line unless contraindicated by heavy alcohol use and advanced liver or kidney disease; it is typically used at 10 to 20 mg weekly with daily folic acid. Dose adjustment for MTX is recommended when estimated glomerular filtration rate less than 60 mL/min/1.73 m^2 and avoided altogether when less than 30 mL/min/1.73 m^2.[5]

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Figure. Approach to Joint Pain in Older Adults

ANA indicates antinuclear antigen; CCP, cyclic citrullinated peptide; CPPD, calcium pyrophosphate deposition; CRP, C-reactive protein; dsDNA, double-stranded DNA; ENA, extranuclear antigen; ESR, erythrocyte sedimentation rate; HbA_{1c}, hemoglobin A_{1c}; HLA, human leukocyte antigen; IA, inflammatory arthritis; IBD, inflammatory bowel disease; ILD, interstitial lung disease; MRI, magnetic resonance imaging; MSU, monosodium urate; RF, rheumatoid factor; RNP, ribonucleoprotein; RS3PE, remitting seronegative symmetrical synovitis with pitting edema; SPEP, serum protein electrophoresis; SSA, Sjögren syndrome antigen A; SSB, Sjögren syndrome antigen B; TSH, thyroid-stimulating hormone; WBC, white blood cell (cells/μL).

[a]Spondyloarthritis includes psoriatic arthritis, ankylosing/axial spondylitis, IBD arthritis, and reactive arthritis. The *HLA-B27* gene is strongly associated but not specific or diagnostic alone.

[b]When to refer to the rheumatology department: IA diagnosis, positive serology results, advanced imaging with synovitis, recurrent relapses, steroid responsiveness, or atypical symptoms.

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Hydroxychloroquine, which is limited to 5 mg/kg/d or less (maximum: 400 mg, daily), is well tolerated among older adults and requires annual eye examinations to monitor retinal toxic effects. Other DMARD agents include sulfasalazine, azathioprine, and leflunomide, which, along with MTX, require blood cell counts and metabolic laboratory monitoring every 3 to 6 months.

Biologic and targeted synthetic DMARDs typically warrant rheumatology referral and careful risk-benefit assessment. Prior authorization and out-of-pocket costs may also delay and affect agent selection.

Glucocorticoids can rapidly reduce inflammation and improve symptoms; however, their use should be limited, especially in older adults. Even at lower doses (eg, prednisone, <5 mg/d), risks of infection, osteoporosis, diabetes, hypertension, mood changes, delirium, and impaired wound healing are increased. If glucocorticoids are used, the lowest effective dose and shortest duration should be prescribed, ideally as temporary bridging therapy, with a plan to transition to a steroid-sparing DMARD.[6] Clinicians should attempt to reduce the glucocorticoid dose by approximately 25% to 50% per week. If ongoing glucocorticoid therapy is required, the dose should be tapered to 5 mg/d or less of prednisone (or equivalent) whenever possible to minimize risks associated with long-term use. For individuals receiving glucocorticoids beyond 3 months, clinicians should address bone protection, vaccination, metabolic monitoring, and provide counseling regarding infection risk.

Additional guidance on diagnosis and prescribing can be found at the American College of Rheumatology's *Rheumatology for Primary Care* resource.[5] Multimorbidity, polypharmacy, mentation, functional limitations, and patient preferences all are associated with IA medication selection and tolerance, underscoring the importance of interdisciplinary care.[7]

Conclusions

IA in older adults presents unique diagnostic and therapeutic challenges. Atypical and overlapping presentations, the coexistence of OA, and vulnerability to treatments can complicate care. By maintaining clinical vigilance, recognizing inflammatory patterns, limiting glucocorticoid exposure, and initiating DMARD therapy promptly, primary care clinicians can reduce diagnostic delay and potentially prevent avoidable functional decline and pain.

ARTICLE INFORMATION

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