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# DIFFERENTIAL DIAGNOSIS OF JOINT SYNDROMES IN OUTPATIENT INTERNAL MEDICINE PRACTICE

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## Abstract

Joint pain and swelling occur in inflammatory, degenerative, crystal-induced, infectious, traumatic, endocrine, and systemic disorders; consequently, this symptom complex requires a structured differential diagnostic approach from the initial outpatient encounter. This article aims to summarize the diagnostic features of joint syndromes and present a clinically applicable initial assessment algorithm based on educational presentation materials and current clinical guidelines. A narrative analysis was conducted of the educational material and recommendations issued by EULAR, NICE, ACR, and EBJIS concerning early arthritis, rheumatoid arthritis, osteoarthritis, spondyloarthritis, gout, crystal-induced arthropathies, and septic arthritis. The synthesis indicated that the initial assessment should begin by excluding emergencies, confirming an intra-articular source of symptoms, and distinguishing inflammatory from mechanical pain patterns. The most informative elements include the combined findings from history taking, the distribution and number of affected joints, duration of morning stiffness, systemic and extra-articular features, and appropriately targeted laboratory and imaging investigations. Normal acute-phase reactants or negative rheumatoid factor and anti-cyclic citrullinated peptide antibodies do not exclude early inflammatory arthritis and should not delay referral to a rheumatologist. This article is a clinical review rather than a study of a patient sample; the source material contained no quantitative findings from a clinical cohort. The proposed algorithm is intended to support, not replace, clinical judgment and specialist consultation.

**Keywords:** joint syndrome; arthritis; arthralgia; differential diagnosis; early arthritis; synovitis; primary health care; rheumatology.

## 1. Introduction

The term joint syndrome encompasses symptoms and objective findings arising from disorders of the joints, synovial membrane, periarticular tissues, and, occasionally, other musculoskeletal structures. In outpatient practice, patients commonly present without an established diagnosis, reporting pain, stiffness, swelling, or restricted movement. These manifestations are nonspecific: similar localized pain may result from inflammatory arthritis, osteoarthritis, crystal deposition, infection, tendon injury, trauma, referred pain, or systemic disease. Thus, “joint syndrome” describes a clinical problem but does not, in itself, identify its etiology. The primary care clinician must determine the urgency of

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assessment, anatomical site of involvement, probable pathophysiological mechanism, and appropriate level of subsequent care. This approach is particularly important early in the disease course, when the characteristic phenotype may not yet be apparent, laboratory findings may be nonspecific, and radiographic abnormalities may be absent [1, 2].

The practical value of recognizing inflammatory arthritis early lies in timely referral to a rheumatologist and avoidance of prolonged, ineffective symptomatic management. EULAR recommendations for early arthritis emphasize prompt specialist assessment when objective joint swelling is present, suggesting rheumatological evaluation within six weeks of symptom onset; NICE recommends urgent referral for suspected persistent synovitis of undetermined cause, particularly when small joints of the hands or feet or multiple joints are involved or when presentation has been delayed [2, 3]. These time frames are organizational targets rather than grounds for postponing assessment in a rapidly deteriorating patient. Early diagnosis matters not because every painful episode warrants disease-modifying treatment, but because persistent inflammation can cause structural damage and functional impairment, whereas the absence of objective inflammation calls for investigation of alternative causes and helps prevent unjustified immunomodulatory therapy.

Joint pain acquires diagnostic significance only when interpreted alongside the medical history and physical examination. The clinician should establish when symptoms began, whether they reached peak intensity within hours or developed gradually, whether similar episodes have occurred, how many joints are involved, and whether their distribution has changed over time. Patients may describe aching, pulling, swelling, or an inability to bend a limb without distinguishing joint-line pain from tenderness of a tendon, bursa, or muscle. Such complaints should therefore be clarified by questions about movement, rest, nocturnal awakening, morning stiffness, physical activity, and everyday function. It is equally important to determine whether restricted movement reflects pain, effusion, a true contracture, muscle weakness, or mechanical obstruction. Bilateral examination can reveal subtle asymmetry, allow comparison of range of motion, and reduce the risk of attributing incidental tenderness to a particular disease.

The differential diagnosis is broad and extends beyond rheumatological conditions. It includes rheumatoid and psoriatic arthritis, axial and peripheral spondyloarthritis, systemic lupus erythematosus, Sjögren syndrome, systemic vasculitis, gout, calcium pyrophosphate crystal arthritis, osteoarthritis, infectious arthritis, reactive arthritis, trauma, and periarticular syndromes. In certain clinical circumstances, joint symptoms may accompany thyroid and metabolic disorders, chronic infections, hematological conditions, or malignancy. The concurrence of symptoms with a known disease does not establish causality: osteoarthritis and inflammatory arthritis, gout and septic arthritis, or fibromyalgia and structural joint damage may coexist in the same patient. Clinicians should maintain several working hypotheses, prioritize them according to likelihood and potential harm, and test them against subsequent findings rather than allowing a single positive laboratory result to replace clinical reasoning [1, 2, 6].

Distinguishing intra-articular inflammation from periarticular pathology is particularly important. Arthritis typically presents with swelling, effusion, joint-line tenderness, and limitation of both active and passive movement. Periarticular pain is more often localized and reproduced by a particular movement or resisted action, while passive

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range of motion may remain relatively preserved. These clinical boundaries are not absolute, however: deep-seated joints, severe pain, obesity, coexisting neuropathy, and chronic disease may complicate the examination. Bursitis, tenosynovitis, and enthesitis can produce appreciable swelling and morning discomfort, whereas early synovitis may occur without conspicuous erythema. Consequently, no single characteristic constitutes a diagnostic test; rather, these distinctions help determine which structure requires further assessment and whether ultrasonography, aspiration, or other imaging is indicated.

Urgent assessment is required for an acutely hot, markedly painful, swollen joint, particularly in the presence of fever, chills, severe malaise, immunodeficiency, recent surgery or injection, a joint prosthesis, skin infection, or risk factors for bacteremia. Septic arthritis cannot be excluded solely because fever is absent, C-reactive protein is normal, or the peripheral leukocyte count is only moderately elevated. Clinical suspicion warrants urgent referral and synovial fluid analysis; the identification of crystals does not exclude concomitant bacterial infection [6]. Other warning signs requiring expedited assessment include recent significant trauma, inability to bear weight, rapidly progressive weakness, acute monoarthritis in an older adult, suspected malignancy, and features of giant cell arteritis in a patient with polymyalgia symptoms, including new-onset headache, visual disturbance, or jaw claudication. Such findings change the order of clinical priorities: potentially destructive or vision-threatening conditions must first be excluded safely to prevent joint damage, systemic complications, or visual loss.

The aim of this article is to present a structured algorithm for assessing joint syndromes in outpatient internal medicine and demonstrate how clinical findings guide investigations and patient referral. The approach draws on the diagnostic principles presented in the educational material “Joint Syndrome in Outpatient Internal Medicine Practice” [1]; however, selected statements have been aligned with current international recommendations, with qualifications added where the original slides might otherwise suggest absolute rules. This review does not estimate the prevalence of particular diagnoses or evaluate the proposed algorithm in a clinical cohort. Instead, it integrates five practical stages: history taking, anatomical localization, identification of an inflammatory pattern, assessment for systemic features, and targeted laboratory or imaging confirmation of the working hypothesis. This framework supports sequential decision-making in primary care, helps explain the rationale for referral to patients, and facilitates continuity of care with rheumatologists, orthopedic surgeons, infectious disease specialists, and other professionals.

## 2. Materials and Methods

This work was undertaken as a narrative clinical review and methodological synthesis. The principal source was an educational presentation entitled “Joint Syndrome in Outpatient Internal Medicine Practice,” which describes the symptom complex, stages of differential diagnosis, features of intra-articular and periarticular involvement, characteristics of inflammatory and mechanical pain, systemic manifestations, and available laboratory and imaging methods [1]. The presentation did not describe a study population, patient inclusion protocol, primary observations, control group, statistical significance testing, or a complete list of references supporting all numerical statements. Accordingly, information from the slides was treated as educational clinical guidance, not as findings from an observational study conducted by the authors.

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To update and verify the clinical recommendations, we consulted EULAR guidance on early arthritis and gout diagnosis, NICE guidance on rheumatoid arthritis, osteoarthritis, and spondyloarthritis, ACR recommendations on gout management, EBJIS guidance on septic arthritis of native joints, the consensus classification criteria for rheumatoid arthritis, and EULAR recommendations on imaging in crystal-induced arthropathies [2–10]. Searches were conducted in PubMed and PubMed Central and on the NICE, EULAR, ACR, and EBJIS websites using document titles and the terms “early arthritis,” “persistent synovitis,” “septic arthritis,” “gout diagnosis,” “osteoarthritis diagnosis,” “spondyloarthritis referral,” “joint aspiration,” and “joint imaging.” Priority was given to guidelines and consensus statements offering actionable recommendations on diagnosis and referral.

The material was analyzed thematically. First, clinical features were organized according to the stages of the assessment algorithm: urgency and red flags, medical history, intra-articular versus periarticular localization, inflammatory versus mechanical pattern, joint distribution, and extra-articular manifestations. Each diagnostic feature was then linked to possible hypotheses and limitations of interpretation. For laboratory investigations, particular attention was paid to whether the test addressed a specific clinical question, whether its result could change management, and whether a negative finding might provide false reassurance. For imaging, we considered the respective roles of radiography, ultrasonography, magnetic resonance imaging, and computed tomography, together with the risk of incidental findings that do not explain symptoms. The tables and diagrams were designed as practical summary aids, not as validated scales, diagnostic criteria, or prognostic models.

Because this review involved no interventions, identifiable medical data, or analysis of individual patient records, local ethics committee approval was not required for its preparation. No registered systematic search protocol, independent study selection by two reviewers, or formal risk-of-bias assessment was undertaken, and no meta-analysis was performed. To avoid overinterpretation, diagnostic features are presented as indicators that modify the probability of a hypothesis rather than establish a diagnosis in isolation. Where guidelines specify urgent referral or a necessary confirmatory procedure, this is stated explicitly. Numerical estimates in the slides that lacked verifiable citations or descriptions of their original sources were not reproduced as established frequencies or measures of diagnostic accuracy. The conclusions concern clinical reasoning and referral of adult outpatients and do not supersede local protocols, the treating clinician’s decisions, or specialist consultation.

## 3. Results

The synthesis of the sources confirmed that a joint syndrome should be approached as a sequence of diagnostic decisions rather than a checklist of laboratory orders. The first decision concerns patient safety: can outpatient assessment proceed, or is emergency care required? The second is whether there is objective intra-articular inflammation, periarticular pathology, or pain without identifiable local abnormalities. The third concerns the predominant pattern: acute or chronic, monoarticular or polyarticular, inflammatory or predominantly mechanical. The fourth is whether the skin, eyes, mucous membranes, gastrointestinal or genitourinary systems, lungs, heart, kidneys, nervous system, or muscles are involved. Laboratory investigations and imaging should be selected only after these

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questions have been addressed. This sequence reduces indiscriminate ordering of extensive test panels and facilitates interpretation of each result within the context of a preliminary clinical hypothesis [1–3, 6].

A comprehensive history should address not only the nature of the pain but also its temporal pattern, preceding infections, trauma, physical loading, operations, intra-articular procedures, and medication use. The clinician should distinguish acute onset over hours or days, subacute progression, gradual worsening over weeks, and recurrent episodes spanning months. For each affected site, it is important to establish whether swelling was visible, redness or increased skin temperature occurred, weight-bearing was impaired, pain caused nocturnal awakening, and stiffness persisted after sleep or rest. An association with recent pharyngitis, diarrhea, urethritis, skin infection, a tick bite, travel to an endemic region, a new medication, or dose adjustment may substantially alter the differential diagnosis. A history of psoriasis, inflammatory bowel disease, uveitis, or familial spondyloarthritis is particularly informative in patients with chronic back pain or enthesitis [5].

Age, sex, occupation, and lifestyle modify the prior probability of individual diagnoses but cannot replace examination. Gout is more common among middle-aged and older men, although it also occurs in women and at atypical sites; rheumatoid arthritis is diagnosed more frequently in women, but male sex does not exclude the disease. Mechanical loading, repetitive movements, occupational microtrauma, sport, excess body weight, and previous injuries may aggravate degenerative and periarticular symptoms. In younger patients, appropriate risk factors increase suspicion of spondyloarthritis, reactive arthritis, and infectious causes, whereas new-onset acute arthritis in an older patient requires careful assessment for infection, crystal-induced arthritis, medication-related causes, and malignancy. These associations are useful when generating hypotheses, but they overlap substantially in clinical practice; accordingly, age- and sex-related trends are not presented here as rules with high diagnostic accuracy [2, 5–7].

During examination, the clinician compares the affected joint with its contralateral counterpart and assesses contours, effusion, local temperature, tenderness, active and passive motion, stability, crepitus, limb alignment, weight-bearing, and functional ability. In the hands and feet, the distribution of swelling, the number of affected joints, and involvement of the proximal interphalangeal, metacarpophalangeal, distal interphalangeal, and wrist joints are important. The examination should extend beyond the site indicated by the patient: Achilles tendon and plantar fascia enthesitis, dactylitis, inflammation of tendon sheaths, bursal tenderness, and proximal muscle stiffness may suggest a systemic disorder. Both active and passive range of motion should be assessed; marked restriction of both favors an intra-articular source, whereas pain elicited by one specific movement or resistance test more often points to periarticular structures. No individual finding is sufficiently specific to establish a definitive diagnosis [1, 6].

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**Table 1. Clinical Features of Intra-Articular and Periarticular Involvement**

| Feature | Likely intra-articular involvement | Likely periarticular involvement |
|---|---|---|
| Pain location | Diffuse pain or joint-line tenderness | Localized tenderness over a tendon, enthesis, or bursa |
| Symptom provocation | Pain may occur at rest or with movement; tenderness in several directions of motion | Pain is more often reproduced by a particular movement, stretch, or resisted action |
| Active and passive movements | Both active and passive motion may be limited in multiple planes in synovitis or effusion | Active movement is more restricted; passive range of motion may be preserved |
| Swelling | Effusion, diffuse enlargement of joint contour, synovial hypertrophy | Localized swelling along a tendon or over a bursa; occasionally linear |
| Further investigation | Ultrasonography if effusion is uncertain; aspiration for unexplained inflammatory monoarthritis | Targeted ultrasonography for suspected tenosynovitis, bursitis, or enthesitis |

Table 1 compares intra-articular and periarticular involvement. In synovitis, tenderness may be diffuse, extend along the joint line, and accompany effusion or restricted motion in several planes. In tendinopathy, tenosynovitis, enthesitis, or bursitis, pain is more commonly localized to the affected anatomical structure and reproduced by loading, stretching, or resisted movement. Nevertheless, localized tenderness does not exclude synovitis, and periarticular inflammation may coexist with intra-articular disease. If examination cannot reliably establish whether effusion or synovial thickening is present, ultrasonography can clarify the anatomical source and assist in selecting an aspiration site. Conversely, routine MRI for every localized pain complaint is unnecessary: imaging should answer a specific question that cannot be resolved clinically or through a more accessible technique. Imaging findings must always be correlated with symptoms, as structural abnormalities may also occur in individuals without current pain.

A central clinical distinction is the likelihood of inflammation. An inflammatory pattern is more commonly associated with prolonged morning stiffness, pain at rest or during the night, improvement with movement, swelling, and restricted motion in multiple planes. Systemic features such as fatigue, low-grade fever, reduced appetite, weight loss, or elevated acute-phase reactants may support this hypothesis but are not present in every form of inflammatory arthritis. Mechanical pain typically worsens with activity or toward the end of the day and improves with rest; morning stiffness is generally brief. The terms “inflammatory” and “mechanical” describe clinical tendencies rather than mutually exclusive biological categories: advanced osteoarthritis may cause nocturnal pain and effusion, while patients with chronic arthritis may develop secondary mechanical pain.

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Therefore, pain patterns should be interpreted alongside examination findings and clinical evolution, rather than used as a single test to determine treatment [2–5].

The duration of morning stiffness helps direct the diagnostic assessment but is not a rigid threshold for any diagnosis. Stiffness lasting more than one hour increases suspicion of active inflammation, especially when accompanied by symmetrical small-joint synovitis and tenderness on passive movement. Stiffness lasting up to 30 minutes, together with activity-related pain and age over 45 years, supports a clinical diagnosis of osteoarthritis; NICE notes that typical osteoarthritis in adults can often be diagnosed clinically without routine imaging [4]. Prolonged immobility may aggravate stiffness in other conditions, while sedatives, depression, sleep disorders, and coexisting fibromyalgia affect subjective reporting. It is important to document not only the duration in minutes but also its functional impact: can the patient make a fist, dress, rise from a chair, climb stairs, and how long does it take for movement to feel normal? Reassessment over time is often more informative than a single approximate estimate.

The rate of symptom development, severity, attack duration, and recurrence provide another diagnostic dimension. A sudden episode of severe pain with swelling and erythema in a single joint may indicate gout or calcium pyrophosphate crystal arthritis, but infectious arthritis must remain in the differential diagnosis until sufficient evidence is obtained. Subacute onset involving several inflamed joints increases the likelihood of early rheumatoid, viral, or reactive arthritis, whereas gradually progressive activity-related pain is more typical of osteoarthritis. Episodic symptoms may occur in crystal-induced arthropathy, but recurrence alone does not establish gout without supporting clinical or microscopic evidence. When pain is continuous and progressive, clinicians should investigate infectious, neoplastic, and systemic causes rather than repeatedly prescribing analgesics. A clear symptom timeline is particularly useful when the patient presents after an acute episode has subsided and the examination no longer reveals marked inflammation.

The number and symmetry of affected joints narrow, but do not determine, the differential diagnosis. Monoarthritis requires priority exclusion of infection, crystal-induced arthritis, trauma, and intra-articular injury. Lower-limb oligoarthritis may occur in spondyloarthritis, reactive arthritis, gout, and psoriatic arthritis. Persistent symmetrical polyarthritis of the hands and feet increases the probability of rheumatoid arthritis but may also occur in viral infections, systemic lupus erythematosus, drug-induced disease, and other connective tissue disorders. Distal interphalangeal joint involvement with psoriatic plaques, nail changes, or dactylitis supports psoriatic arthritis, whereas Heberden and Bouchard nodes in the setting of mechanical loading and age-related changes are characteristic of hand osteoarthritis. Gout frequently begins in the first metatarsophalangeal joint but may later become polyarticular. Thus, simplified teaching models linking a single diagnosis to a single joint distribution are useful only as preliminary guides [1, 2, 6].

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**Table 2. Joint Involvement Patterns and Diagnostic Hypotheses**

| Clinical presentation | Possible diagnoses | Limitations of interpretation |
|---|---|---|
| Acute hot monoarthritis | Septic arthritis, gout, calcium pyrophosphate crystal arthritis, trauma | Infection must be excluded urgently; crystals do not rule out concurrent infection |
| Symmetrical small-joint synovitis of the hands or feet | Rheumatoid arthritis; also viral and systemic diseases | Negative RF or anti-CCP does not exclude early or seronegative arthritis |
| Distal joint involvement, dactylitis, psoriasis, or nail changes | Psoriatic arthritis | Skin findings may be hidden; the joint pattern is variable |
| Asymmetrical lower-limb oligoarthritis, enthesitis | Spondyloarthritis, reactive arthritis, crystal-induced arthritis | Assess psoriasis, uveitis, intestinal or genitourinary infection, and axial pain |
| Activity-related pain, brief stiffness, osteophytes or nodes | Osteoarthritis | Diagnosis is often clinical in typical cases; imaging for atypical presentations |
| Shoulder and pelvic girdle pain with prolonged stiffness in an older adult | Polymyalgia rheumatica; inflammatory myopathy and other causes | Assess muscle strength and signs of giant cell arteritis |

The combination of joint manifestations with cutaneous, ocular, and mucosal findings may be more informative than an isolated laboratory test. Psoriatic plaques, nail changes, and dactylitis support psoriatic arthritis; photosensitive rash, mucosal ulcers, cytopenias, proteinuria, and characteristic immunological abnormalities warrant evaluation for systemic lupus erythematosus. Xerostomia and xerophthalmia may accompany Sjögren syndrome, although medications, dehydration, diabetes, and aging can also cause dryness. Conjunctivitis or anterior uveitis combined with peripheral arthritis, inflammatory back pain, or a preceding genitourinary or intestinal infection suggests spondyloarthritis or reactive arthritis. Nodules, tophi, Raynaud phenomenon, skin thickening, purpura, ulcers, or livedo warrant a targeted systemic examination. These associations do not themselves constitute diagnoses: skin lesions may be incidental, and uncertain morphology should be documented by site and, where appropriate, reviewed by a dermatologist [5, 10].

Examination of lymph nodes, liver, spleen, lungs, and heart is relevant when systemic symptoms are present or the joint findings are inconsistent with a presumed localized diagnosis. Generalized lymphadenopathy, splenomegaly, cytopenias, persistent fever, and rash may accompany systemic inflammatory disorders but also require evaluation for infectious and hematological causes. Pleuritis, interstitial changes, pericarditis, glomerular involvement, and neuropathy alter diagnostic priorities and may indicate systemic disease. An older patient presenting with bilateral shoulder and pelvic

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girdle pain, morning stiffness, elevated inflammatory markers, and reduced general activity should be assessed for polymyalgia rheumatica; a new headache, scalp tenderness, transient visual loss, or jaw claudication requires immediate exclusion of giant cell arteritis. Myalgia should not automatically be classified as arthritis: muscle strength, proximal function, creatine kinase, and medication history should be evaluated when myopathy is suspected.

Paraneoplastic joint manifestations are uncommon compared with degenerative and prevalent inflammatory disorders; therefore, arthralgia alone does not justify routine cancer screening beyond established indications. Greater vigilance is warranted with new-onset inflammatory symptoms in older age, unusually rapid progression of polyarthralgia, asymmetric oligoarthritis of large joints, unexplained weight loss, night sweats, persistent fever, anemia, lymphadenopathy, bleeding, or focal bone pain. Decisions regarding investigation should integrate the clinical presentation, risk factors, and established age-appropriate cancer screening programs. Two extremes should be avoided: overlooking systemic warning signs and indiscriminately ordering extensive imaging or tumor-marker panels. If inflammatory arthritis remains a plausible diagnosis, cancer risk does not negate the need for rheumatology referral; these diagnostic pathways can proceed in parallel. The original presentation emphasizes oncological awareness, but its numerical estimate for paraneoplastic arthritis was not incorporated into this review's conclusions because the primary source could not be verified [1].

Laboratory testing should evaluate a clearly formulated clinical hypothesis and assess systemic risks. A complete blood count can identify anemia, leukocytosis, leukopenia, and thrombocytopenia; erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) help assess inflammatory activity and trends but are not specific for rheumatological disease. Biochemical testing may include liver and renal function before potential treatment, albumin, electrolytes, creatine kinase if myopathy is suspected, and serum urate when gout is clinically suspected. Urinalysis and the urine protein-to-creatinine ratio are appropriate when systemic disease or renal involvement is suspected. Rheumatoid factor (RF) and antibodies to citrullinated peptides are relevant when clinical synovitis suggests rheumatoid arthritis; antinuclear antibodies are indicated when features suggest systemic connective tissue disease. Ordering all these tests for every patient increases false-positive findings, repeat investigations, and diagnostic anchoring. Each test should address a specific question, and negative results must be interpreted in the context of pretest probability [2, 3, 10].

Rheumatoid factor and antibodies to cyclic citrullinated peptides (anti-CCP) have diagnostic and prognostic value, but neither can replace clinical assessment. RF may be present in other autoimmune diseases, chronic infections, older individuals, and occasionally people without clinical rheumatoid arthritis. Anti-CCP antibodies are more specific, yet a negative result does not exclude seronegative or early disease. The 2010 ACR/EULAR classification criteria were developed to classify patients with established inflammatory joint involvement and should not be applied mechanically as a diagnostic gatekeeping tool in primary care [10]. NICE recommends RF testing in patients with clinical synovitis, consideration of anti-CCP testing when RF is negative, and referral without waiting for laboratory results [3]. Serology can therefore support clinical decisions,

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inform assessment of the risk of persistent or erosive disease, and assist the rheumatologist; however, it should not be used to dismiss a diagnosis when clinical findings are compelling.

Serum urate concentrations require particularly cautious interpretation. Hyperuricemia increases the likelihood of gout but also occurs in people with no clinical attacks; concentrations may be normal during an acute flare. A diagnosis should therefore neither be established solely on one elevated value nor excluded on the basis of a normal result during an attack. When feasible and clinically indicated, identification of monosodium urate crystals in synovial fluid or tophus aspirate remains the reference standard for confirmation. EULAR recommends synovial fluid analysis in suspected gout and undifferentiated inflammatory arthritis; when this is unavailable, clinical findings and, if uncertainty persists, imaging should be used [7]. This is particularly important in atypical presentations, older patients, polyarticular involvement, or suspected infection. Radiography may demonstrate chronic structural changes, but normal early radiographs do not exclude gout or another inflammatory disorder.

Joint aspiration is both a diagnostic tool and a critical step when septic arthritis is suspected. Synovial fluid should be submitted for microbiological assessment, leukocyte count with differential, and crystal analysis; if the sample volume is limited, test priorities should be determined according to clinical risk in consultation with the laboratory [6]. A synovial leukocyte count exceeding 50,000 cells/µL increases suspicion of infection but does not independently confirm it; a lower count reduces the probability without excluding septic arthritis, particularly in immunosuppressed patients or after antibiotic treatment. The presence of crystals does not rule out concurrent infection. In a hemodynamically stable patient, diagnostic material should be obtained before initiating antibiotics, provided this does not create a dangerous delay; patients with signs of sepsis require immediate management according to local protocols. Aspiration must be performed by trained personnel using aseptic technique, and where the procedure is unavailable, the patient should be referred urgently to an appropriate facility.

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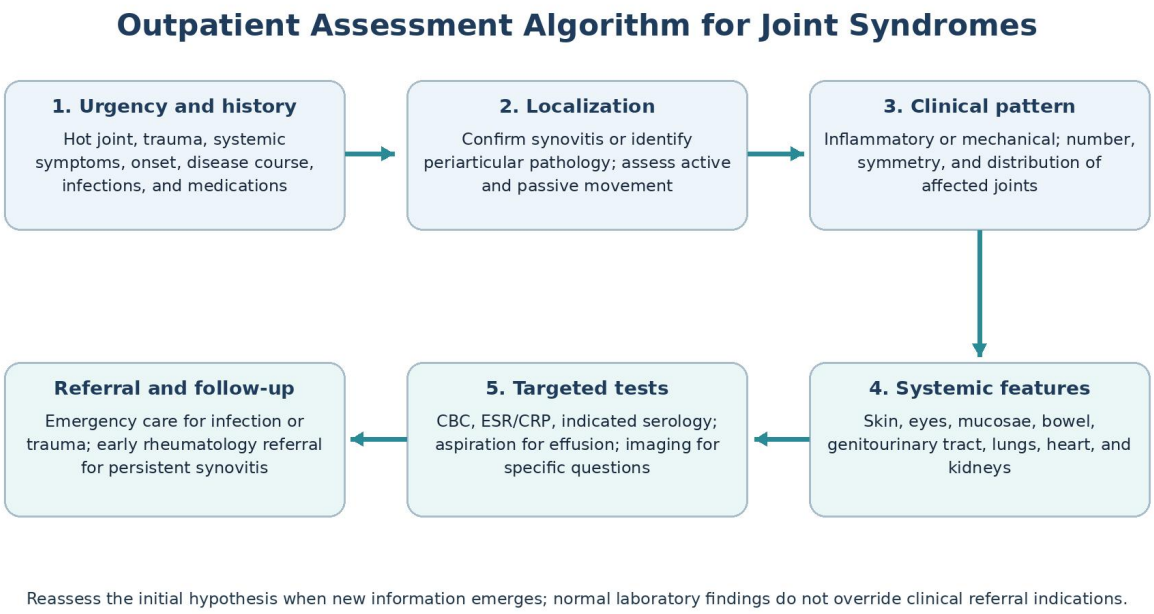



*Figure 1. Sequential outpatient assessment of joint syndromes. The algorithm is based on the source presentation and recommendations [1–6].*

Imaging should be selected to address a specific clinical question. Radiography can detect fractures, structural damage, erosions, joint-space narrowing, osteophytes, chondrocalcinosis, and late-stage changes, but may be normal early in inflammatory disease. When diagnostic uncertainty persists, ultrasonography or MRI may complement clinical examination, although their findings must also be interpreted clinically [11]. For a typical presentation of osteoarthritis in adults, NICE does not recommend routine imaging to confirm the diagnosis; imaging is particularly useful when features are atypical, an alternative diagnosis or complication is suspected, or surgery is being considered [4]. Ultrasonography can identify effusion, synovial hypertrophy, tenosynovitis, tendinitis, enthesitis, bursitis, and crystal deposits and can guide aspiration. Ultrasonography is operator-dependent and should answer the clinical question posed; minimal subclinical abnormalities should not automatically be regarded as the source of pain. In EULAR recommendations for early arthritis, clinical examination remains the primary method for detecting arthritis, while ultrasonography can help confirm equivocal synovial findings [2].

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**Table 3. Extra-Articular Features Guiding Diagnostic Assessment**

| Examination site | Example finding | Further assessment |
|---|---|---|
| Skin and nails | Psoriatic plaques, nail dystrophy, Raynaud phenomenon, purpura | Psoriatic arthritis, systemic connective tissue disease, or vasculitis |
| Eyes | Acute painful red eye, photophobia, reduced vision | Urgent ophthalmological assessment if uveitis is suspected |
| Mucous membranes | Oral ulcers, marked dryness of eyes and mouth | Systemic connective tissue disease; review medications and alternative causes |
| Gastrointestinal and genitourinary systems | Recent diarrhea, urethritis, inflammatory bowel disease | Reactive arthritis or spondyloarthritis in a compatible clinical phenotype |
| Lymph nodes and organs | Lymphadenopathy, splenomegaly, pulmonary or cardiac features | Systemic inflammatory, infectious, or hematological disease |
| General symptoms | Fever, weight loss, night sweats, new headache, or visual symptoms | Exclude infection, systemic disease, and other urgent causes |

MRI offers advantages for evaluating bone marrow, soft tissues, deep joints, early sacroiliitis, and certain occult injuries, but accessibility, cost, and incidental findings limit its role as a first-line test for every joint complaint. Computed tomography (CT) provides detailed assessment of bone structure, complex fractures, and certain crystal deposits, including dual-energy CT when the diagnosis of gout remains uncertain, but is not necessary for all patients. Chest radiography, electrocardiography, echocardiography, abdominal and renal ultrasonography, electroneuromyography, and biopsy should be ordered according to a specific hypothesis regarding systemic involvement, rather than routinely for every patient labeled as having “arthritis.” EULAR recommends ultrasonography and dual-energy CT in appropriate diagnostic circumstances for crystal-induced arthropathies; radiography and ultrasonography are also relevant for chronic calcium pyrophosphate crystal disease [9]. Choice of modality should take into account whether the result will change management, whether a more accessible investigation is available, and the risks associated with delayed referral.

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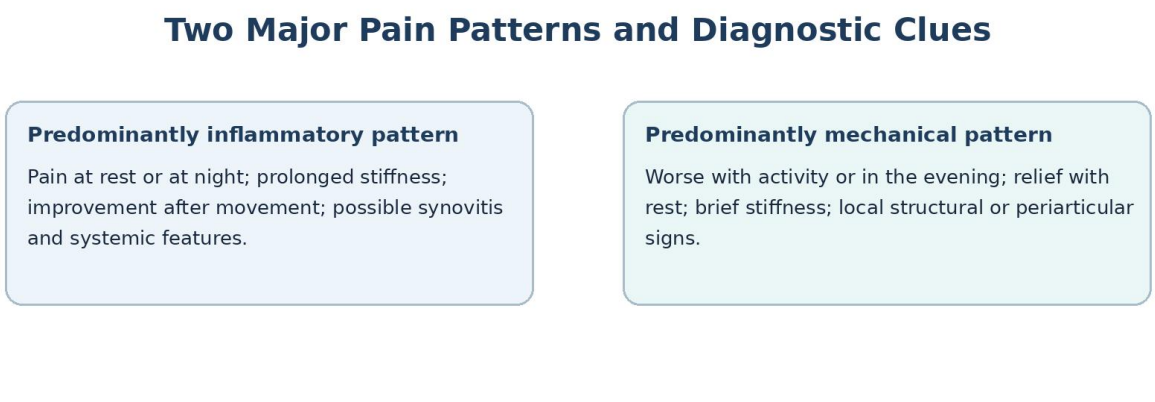



*Figure 2. Comparison of predominantly inflammatory and mechanical pain patterns. These findings are indicative and should be assessed together [2–5].*

The summary in Table 2 brings together joint involvement patterns and diagnostic hypotheses but is intended to guide assessment, not establish a diagnosis based on one matching feature. Rapidly developing monoarthritis of the foot with recurrent attacks and erythema increases the probability of gout; crystal identification or characteristic imaging findings can support confirmation. Symmetrical synovitis affecting the wrists, metacarpophalangeal joints, and proximal interphalangeal joints increases the likelihood of rheumatoid arthritis, although diagnosis still requires clinical confirmation, serology, and consideration of alternative causes. Psoriasis, nail dystrophy, and dactylitis suggest psoriatic arthritis, whereas inflammatory back pain, uveitis, enthesitis, or inflammatory bowel disease suggest spondyloarthritis. Activity-related pain in the knee or hip accompanied by brief morning stiffness supports osteoarthritis but does not exclude effusion or concurrent inflammation. This framework helps formulate a differential diagnosis before test results are available and reduces the risk of one laboratory abnormality overshadowing the overall clinical picture.

The systemic features listed in Table 3 demonstrate the diagnostic value of a targeted examination. Mucosal ulcers, photosensitive rash, Raynaud phenomenon, proteinuria, and cytopenias warrant investigation for systemic connective tissue disease. Dry eyes and mouth require review of medications and objective findings before a diagnosis of Sjögren syndrome is made. Conjunctivitis, and particularly a painful red eye with photophobia, should not be dismissed as an “associated symptom”: suspected anterior uveitis requires urgent ophthalmological assessment. Psoriatic changes of the skin or nails may go unnoticed by patients; therefore, inspection of the scalp, umbilicus, intergluteal cleft, and nails is important when psoriatic arthritis is possible. Lymphadenopathy, hepatosplenomegaly, and pulmonary or cardiac findings do not establish a rheumatological cause of arthritis, but broaden the differential diagnosis and call for coordinated multidisciplinary evaluation. In each case, a working diagnosis should account for both the articular and extra-articular components of the syndrome.

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Rheumatology referral is indicated for persistent objective synovitis of uncertain cause, inflammatory involvement of multiple joints, small-joint involvement of the hands or feet, suspected early rheumatoid arthritis, psoriatic arthritis, spondyloarthritis, systemic connective tissue disease, or recurrent unexplained episodes of arthritis. Negative RF and anti-CCP tests or normal ESR and CRP should not delay referral when clinical suspicion is substantial [2, 3]. An acutely hot joint with possible infection requires urgent assessment for aspiration and exclusion of septic arthritis rather than a routine rheumatology appointment [6]. In patients with features of axial spondyloarthritis, referral may be warranted despite normal sacroiliac radiographs, because early abnormalities may not be visible; NICE provides clinical referral criteria, but diagnosis ultimately integrates symptoms, laboratory results, and imaging [5]. Referral pathways and time frames should reflect symptom severity and regional healthcare resources.

The five stages of differential assessment are summarized in Figure 1. Stage one comprises history taking and risk assessment, including rapid screening for infection, trauma, and systemic threats. Stage two establishes the anatomical site and distinguishes joint pathology from disorders of surrounding tissues. Stage three classifies the clinical pattern according to inflammatory and mechanical characteristics, time course, number of joints, and distribution. Stage four identifies extra-articular manifestations and features of systemic disease. Stage five selects investigations that can confirm a hypothesis, exclude a dangerous alternative, or alter management. The diagram emphasizes the iterative nature of the algorithm: new results may require revisiting the history and reconsidering the initial hypothesis. When symptoms remain unexplained, patients need a follow-up plan and clear instructions regarding deterioration that warrants earlier reassessment. Recording the number of swollen joints, duration of stiffness, functional limitations, and changes in laboratory measures makes subsequent consultations more informative and enables assessment over time.

Medication history is an essential component of assessment because drugs may cause arthralgia, increase susceptibility to infection, alter laboratory parameters, or complicate symptom interpretation. Clinicians should establish when glucocorticoids, immunosuppressants, anticoagulants, diuretics, statins, and recently prescribed antibiotics were started or stopped, and document over-the-counter anti-inflammatory medications and supplements. Glucocorticoids may suppress inflammatory findings and mask fever; immunosuppression can modify the presentation of infection and requires caution when interpreting relatively low synovial fluid cell counts. Some medications affect serum urate, and measurements taken without considering treatment may be misinterpreted. Patients should not discontinue treatment independently on the basis of a suspected association; instead, the temporal relationship should be documented and discussed with the prescribing clinician. A detailed record of current and recently modified medications reduces inappropriate attribution of symptoms to pre-existing chronic disease and helps identify drug-induced causes during subsequent assessment.

Functional assessment makes documentation of joint syndromes more reproducible and helps distinguish changes in pain intensity from deterioration in the ability to perform everyday activities. Patients may be asked about difficulty walking, climbing stairs, rising from a chair, dressing, holding objects, or fulfilling occupational duties. Documenting limitations is useful even before a formal disease activity scale has been selected: it

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provides a baseline, facilitates subsequent comparison, and clarifies which changes the patient considers meaningful. On examination, pain-related limitations should be distinguished from weakness, instability, deformity, fear of movement, and impaired balance. Chronic osteoarthritis pain may reduce function without active synovitis, while inflammatory activity may persist despite moderate pain, especially in patients receiving analgesics. Decisions about referral and additional investigations must therefore incorporate both objective inflammation and functional impact, rather than relying on a single numerical score.

The clinical picture may be incomplete at the initial consultation. A follow-up plan is therefore particularly important when a patient is stable and has no emergency warning signs but the diagnosis remains uncertain. At reassessment, the clinician should determine whether effusion persists, additional joints have become involved, stiffness or function has changed, or rash, ocular symptoms, or signs of infection have appeared. Follow-up must not substitute for urgent referral in persistent synovitis or suspected infection; its purpose is to clarify symptom evolution when the initial examination was insufficiently informative. Patients should be told in advance when reassessment will occur, which symptoms to monitor, and what signs of deterioration require earlier care. This approach reduces the risk of losing patients to follow-up after a single course of symptomatic treatment and helps distinguish self-limited episodes from emerging chronic inflammatory disease. Documentation should specify changes in swelling, movement, the number of affected joints, and temperature rather than merely stating that “pain persists.”

The quality of specialist referral depends on how the information is organized. Referral documentation should include the date and tempo of symptom onset, duration of morning stiffness, joints with objective swelling, presence of effusion, local temperature, restriction of active and passive movement, systemic symptoms, preceding infections or trauma, comorbidities, and medications. Investigation results should be reported with dates and clinical context, not merely as a list of values. When infection is suspected, aspiration and microbiological testing, whether performed or still required, should be specified. Pending laboratory tests do not preclude urgent referral for persistent synovitis [2, 3]. This format supports continuity of care and helps the rheumatologist establish whether the presentation is consistent with early inflammatory arthritis or requires a different diagnostic pathway. Structured documentation is especially valuable where specialist appointments are difficult to obtain or referrals are conducted remotely, because the quality of the initial assessment informs triage priority.

Axial pain should be assessed separately from peripheral arthritis, since patients may describe pain in the buttocks, lower back, or chest as “joint pain.” Features favoring inflammatory back pain include onset at a young age, gradual development, improvement with movement, lack of relief at rest, nocturnal awakening, and alternating buttock pain; uveitis, psoriasis, inflammatory bowel disease, enthesitis, and relevant family history increase the diagnostic weight of this pattern. However, no single feature, including HLA-B27 positivity, confirms the diagnosis without clinical context, and normal sacroiliac radiographs do not exclude early axial spondyloarthritis. NICE recommends considering symptom duration, age at onset, and additional clinical features when deciding on rheumatology referral and selecting imaging according to the suspected stage of disease [5]. Mechanical back pain is common and may coexist with spondyloarthritis; clinicians should

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therefore determine precisely which characteristics and associated manifestations justify further investigation.

**4. Discussion**

This synthesis supports the practical value of a stepwise approach because diagnostic errors often arise not from a lack of sophisticated tests but from asking the right questions in the wrong order. Starting with an extensive laboratory panel may reveal incidental seropositivity or asymptomatic hyperuricemia and lead to premature diagnostic anchoring. Conversely, when examination does not confirm synovitis, joint pain combined with an isolated laboratory abnormality does not provide a sufficient basis for disease-modifying antirheumatic therapy. The opposite error—waiting for a fully developed clinical presentation or all test results in a patient with persistent inflammatory arthritis—may delay specialist care. EULAR and NICE emphasize clinical assessment and prompt referral when objective swelling is present, even if acute-phase reactants or serology are normal [2, 3]. In outpatient practice, diagnostic competence therefore involves not only recognizing a particular disease but also identifying when further evaluation should be transferred to a specialist.

Distinguishing inflammatory from mechanical pain is useful when treated as a probabilistic rather than binary classification. The classic descriptions—nocturnal pain and prolonged stiffness in inflammatory disease versus activity-related pain relieved by rest in degenerative disease—help formulate a working hypothesis and guide examination. In practice, however, patients rarely present with a textbook pattern: osteoarthritis may be accompanied by synovitis, inflammatory arthritis by structural damage and secondary mechanical pain, and prolonged inactivity may worsen stiffness in both conditions. Subjective pain assessment is also influenced by sleep, mood, functional status, and coexisting sensitization. It is therefore equally important to establish which movements or activities provoke symptoms, how pain changes after rest, whether passive movement remains restricted, and whether visible or palpable inflammation is present. This more detailed assessment improves the clinical value of the history and helps explain to patients why the nature of pain alone cannot determine a diagnosis.

Age- and sex-related trends highlighted in the original slides retain educational value but should not become exclusionary diagnostic rules. A man with polyarthritis may have rheumatoid arthritis; a woman with an acute gout-like attack may indeed have gout; and inflammatory shoulder pain in an older patient does not necessarily indicate osteoarthritis. Disease frequencies vary between populations, while sex, age, comorbidities, and access to specialists influence the observed clinical presentation. Sex should not be used as an argument against a less common but potentially serious diagnosis. Similarly, occupational factors and dietary habits may identify mechanical load, trauma, or potential triggers of attacks, but cannot themselves establish causality. It is more reliable to document a specific exposure and its temporal relationship to symptoms and then examine whether the association is supported by a reproducible clinical pattern, laboratory findings, or imaging. Such an approach reduces stereotyped diagnostic reasoning and preserves individualized patient assessment.

A joint involvement pattern may appear compelling to a specialist but is not always stable in early disease. Rheumatoid arthritis may begin in only a few joints or present with unilateral symptoms; gout may affect joints other than the first metatarsophalangeal joint;

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and psoriatic arthritis may precede cutaneous psoriasis. Reactive arthritis may follow an infection that has resolved by the time of assessment, while HLA-B27 alone is not a diagnostic test for spondyloarthritis. Clinical feature tables should therefore generate possible diagnoses and questions for further evaluation, rather than function as decontextualized scoring systems. Repeat examination may reveal extension of joint involvement, newly apparent enthesitis or dactylitis, and persistence of synovitis. When the diagnosis remains uncertain, it is useful to specify in advance a follow-up interval and criteria for earlier reassessment: new swelling, fever, rapidly worsening function, ocular symptoms, or emerging systemic manifestations may require accelerated evaluation.

Acute monoarthritis deserves particular attention because clinically suspected gout and infectious arthritis may present similarly, while delays in treating infection carry substantial risks. Even a classic site of gout does not exclude septic arthritis; fever may be absent, and ESR and CRP cannot reliably rule out infection. In the presence of an effusion of uncertain cause, aspiration provides samples for microbiological and crystal analysis. The SANJO guideline recommends aspiration as soon as possible when infection is suspected and cautions against using any single synovial fluid leukocyte count as an absolute criterion for confirming or excluding septic arthritis [6]. In primary care, this translates into a clearly defined referral pathway: when aspiration cannot be performed locally, the patient should be sent to a suitable department rather than undergo delayed evaluation while receiving repeated courses of analgesics. Even after crystals are detected, the clinician should continue to assess for infection if risk remains, because the two disorders can coexist.

Laboratory interpretation requires an understanding of each marker's limitations. ESR may increase with age, anemia, infection, pregnancy, renal disease, and other factors; CRP reflects inflammation but does not identify its location or cause. Normal values do not exclude early arthritis, particularly when objective swelling persists. RF and anti-CCP testing should be reserved for clinical suspicion of rheumatoid arthritis, and antinuclear antibody testing for appropriate systemic features; broad screening of low-risk patients generates false-positive results and unnecessary subsequent investigations. Serum urate neither establishes the diagnosis of gout nor excludes it during an acute flare [7]. Training in laboratory interpretation should include pretest probability: identical results have different implications in a patient with symmetrical hand synovitis and one with isolated mechanical knee pain. For each test, it is useful to specify in advance which question it should answer and what action would follow a positive, negative, or borderline result.

Imaging likewise should be driven by clinical questions. Radiography is well suited to assessing bone structure, traumatic injury, and late-stage abnormalities, but is not a universal exclusion test for early arthritis. Ultrasonography may detect occult effusion, synovial hypertrophy, and periarticular inflammation, assess vascularity, and guide aspiration; interpretation, however, depends on examiner expertise and the examination protocol. MRI is appropriate when a specific question remains regarding deep structures, bone marrow, sacroiliitis, or occult injury and when the answer could change management or referral. NICE advises against routine imaging in typical osteoarthritis [4], whereas EULAR supports ultrasonography and dual-energy CT in selected cases of crystal-induced arthropathy [9]. Excessive imaging can reveal age-related abnormalities unrelated to symptoms and increase patient anxiety. Clinicians should correlate the anatomical location

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of symptoms with imaging findings rather than treating every structural abnormality as the cause of pain.

Searching for systemic manifestations increases the clinical examination's ability to identify disorders that extend beyond the joints. Cutaneous findings, nail abnormalities, mucosal ulcers, dry eyes or mouth, lymphadenopathy, proteinuria, cytopenias, and pulmonary or cardiac features may justify focused investigations. Conversely, indiscriminate screening for occult systemic disease in every patient with arthralgia leads to unnecessary testing. The preferable approach is a targeted examination followed by evaluation of hypotheses generated from the history and physical findings. For example, isolated dry mouth after starting an anticholinergic medication should not automatically prompt a diagnosis of Sjögren syndrome; conjunctivitis without other relevant features is not synonymous with reactive arthritis; and a rash requires characterization and, if necessary, morphological assessment. When systemic disease is suspected, coordination among the internist, rheumatologist, ophthalmologist, nephrologist, and dermatologist reduces duplicated investigations and facilitates interpretation of a complex phenotype.

Appropriate oncological vigilance requires clinical proportionality. Paraneoplastic rheumatic syndromes are recognized but are not among the most common causes of joint pain, and older age alone does not establish a neoplastic etiology. The clinician should assess newly developed constitutional symptoms, unexplained anemia, lymphadenopathy, progressive nocturnal pain, atypical disease course, a mismatch between symptom severity and local findings, and established risk factors. In such situations, targeted investigation according to clinical guidelines and local referral pathways is appropriate. The original presentation's assertion regarding a particular proportion of arthritis cases attributable to paraneoplastic disease is not reproduced here as a confirmed estimate, because the presentation provides no verifiable bibliographic reference, definition of the study population, or calculation method [1]. This decision illustrates a principle of academic caution: even plausible numerical claims should not be included in scientific writing without an identifiable primary source and context.

For practical implementation in primary care, the algorithm must account for access to rheumatologists, joint aspiration, ultrasonography, and laboratory services. If specialist assessment is delayed, the internist can document objective findings, monitor function and inflammatory markers as clinically indicated, assess symptom evolution, and expedite referral if deterioration occurs. Basic investigations must not become mandatory prerequisites that delay referral when the clinical picture suggests persistent inflammatory arthritis [2, 3]. When septic arthritis is an emergency possibility, a pathway is required that enables timely aspiration and treatment [6]. A clear referral should include symptom onset and characteristics, the number and distribution of swollen joints, duration of stiffness, examination findings, and investigations already performed. This is particularly important in regions with limited rheumatology services, where the quality of the initial clinical description may determine the speed and priority of specialist consultation.

The limitations of this article arise primarily from the nature of its source material. The educational presentation brings together clinical recommendations issued in different years and is not an original research study. Some of its numerical claims and descriptions of laboratory characteristics lack citations; these were therefore not treated as verified estimates of diagnostic accuracy or prevalence. This narrative review was not registered,

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the search was not a comprehensive systematic search, publications were not independently selected by two reviewers, and the risk of bias of individual sources was not formally assessed. The tables and algorithms presented have not undergone external validation in an outpatient population and cannot function as stand-alone diagnostic instruments. Furthermore, international recommendations were developed for healthcare systems with differing referral pathways and specialist availability; local application requires adaptation to national protocols and laboratory capacity. Prospective studies in clearly defined populations, using prespecified referral criteria, time to specialist assessment, accuracy of initial hypotheses, and clinical outcomes, are needed to evaluate the algorithm's practical effectiveness.

The practical value of the proposed framework is that it makes clinical reasoning transparent to both clinician and patient. At the first consultation, it can clarify which findings require immediate exclusion of infection or trauma, why negative serology does not settle the question of early arthritis, why aspiration may be necessary, and why radiographs may not be needed to confirm clinically typical osteoarthritis. Patients are more likely to follow a plan when they understand the purpose and timing of investigations and which symptoms should prompt reassessment. Rheumatology colleagues benefit from concise, structured information rather than only a list of tests: date of onset, number of swollen joints, stiffness pattern, psoriasis or ocular symptoms, preceding infection, and aspiration results. Thus, the algorithm can strengthen both diagnostic consistency and continuity of care. Its effectiveness nevertheless depends on access to urgent assessment, accurate documentation, and a willingness to revise the diagnosis as new information emerges.

## 5. Conclusion

Differential diagnosis of joint syndromes in outpatient practice should follow a sequential assessment of urgency, anatomical localization, inflammatory characteristics, joint distribution, and systemic manifestations. Pain, swelling, stiffness, and restricted movement are nonspecific; they gain diagnostic value only when interpreted in relation to symptom chronology, objective examination, and patient context. The most important primary care decisions are to recognize possible septic arthritis or trauma promptly, avoid missing persistent synovitis, distinguish intra-articular inflammation from periarticular pathology, and arrange specialist referral without waiting for a complete laboratory panel. Normal ESR or CRP, negative RF or anti-CCP antibodies, and normal early radiographs do not exclude inflammatory disease when the clinical presentation is convincing. Conversely, hyperuricemia or incidental imaging abnormalities do not establish the cause of symptoms.

Rational investigation begins with a clinical hypothesis and uses laboratory or imaging tests only when their results are likely to influence subsequent decisions. When infection is suspected, urgent aspiration and microbiological assessment take priority; when inflammatory arthritis is likely, early rheumatological consultation is essential. Imaging is often unnecessary in typical osteoarthritis, whereas ultrasonography may clarify the anatomical site of involvement and guide aspiration when localization is uncertain. The tables and diagrams presented synthesize diagnostic features from educational material and current recommendations, but they are not independently validated criteria. Local prospective studies and adaptation of referral pathways to the realities of primary care are required to establish their applicability.

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