

# Diagnosis and Timely Referral of Patients with Early Inflammatory Joint Disease



## Goal of Newsletter:

To educate primary care physicians about promptly identifying and referring patients with inflammatory joint disease to a rheumatologist for consultation and initiation of treatment and working together with the rheumatologist for follow-up and long-term care.

## Educational Objectives:

Upon completion of this activity, participants should be able to:

1. Identify and differentiate inflammatory joint disease from noninflammatory disease.
2. Describe how to identify extra-articular features that help diagnose inflammatory joint disease syndromes.
3. List the laboratory tests that help to confirm the presence of inflammatory disease and assist in making a specific diagnosis.
4. Identify the importance of early referral to the rheumatologist.

## Accreditation:

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Dr. Leonard Sigal, MD, discloses that he is the Director of Immunology, Clinical Design and Evaluation at the Pharmaceutical Research Institute of the Bristol-Myers Squibb Company.

## Introduction

Joint pain is a common complaint that can occur as a result of a number of different disorders, all having various etiologies and clinical presentations. Some complaints of joint pain are due to an underlying inflammatory process, which may affect a single joint (monoarthritis), a few joints (pauciarthritis or oligoarthritis), or many joints (polyarthritis). Regardless of the specific inflammatory etiology, the joint is painful and/or tender, red, swollen, and it does not move or function normally. Evaluation of this joint pain should be comprised of a step-by-step historical and physical evaluation, judicious use of relevant laboratory tests and careful interpretation of results, and a general appreciation of the important clinical features of the more commonly occurring inflammatory joint diseases encountered in primary care. This understanding will help to ensure an accurate diagnosis and appropriate management of inflammatory joint disease. Thus, the purpose of this newsletter is to review the initial process of evaluating inflammatory joint disease, identify clinical features that will help in making a specific diagnosis, and emphasize the importance of early referral to ensure optimal patient outcomes.

## Molecular Biology of Inflammation

Recent advances in molecular biology have helped to uncover the underlying mechanisms of many inflammatory joint diseases. T cells, B cells, macrophages, and synovial cells all play a pivotal role in the inflammatory process. Cytokines (*cyto* means cell and *kine* means movement—literally a chemical made to make cells move or do something) are responsible for cell activation, differentiation, and communication by means of chemical mediators. The best-studied cytokines include the interleukins (IL) (IL-1, IL-2, and IL-6), tumor necrosis factor  $\alpha$  (TNF- $\alpha$ ), the interferons ( $\alpha$ ,  $\beta$ , and  $\gamma$ ), and B-lymphocyte stimulator (BLyS—also known as BAFF for B-cell activation factor), all of which are current therapeutic targets. Interference of cytokines binding to their cell-surface receptors represents one of the most promising methods of treating rheumatoid arthritis (RA), psoriatic arthritis (PsA), and ankylosing spondylitis (AS). Another attractive strategy is to inhibit the antigen-initiated commencement of immune responses that are orchestrated by cytokines and underlie autoimmune reactivity. These therapies block the interaction of the antigen-presenting cell with the T cell. When stimulated by certain cytokines, macrophages and synovial cells produce a number of molecules that magnify the inflammatory process, including prostaglandins, degradative enzymes such as proteinases and collagenases, toxic radicals (oxygen and nitrogen), and inflammatory cytokines.

## Evaluating Joint Disease

The differential diagnosis of joint disease is almost entirely defined by the patient's history and physical examination. It requires careful documentation of symptoms, functional status, evidence of disease activity, and presence of extra-articular features. Whereas the results of laboratory tests can support a clinical diagnosis, they can also be misleading and incur unnecessary time and cost. The results of laboratory tests contribute far less to the process of making the correct rheumatologic diagnosis than a complete history and physical examination. A systematic approach is needed to characterize joint pain:

- Is the “joint” pain articular or is it really periarticular?
- Is the joint disease inflammatory or noninflammatory?
- What is the pattern (physical and temporal) of the joint disease?
- Are extra-articular features present?

### Articular vs Nonarticular Pain

Articular pain is an indicator of either inflammatory or noninflammatory disease that affects the joint itself. In contrast, periarticular pain is an indicator of dysfunction or inflammation of surrounding tissues and structures, including conditions such as bursitis, tendonitis, and cellulitis. One must differentiate articular from periarticular pain by identifying the exact site of pain, factors that exacerbate or improve pain, and range of motion. The patient's own finger—denoting the precise location of pain—is the best diagnostic tool, and it may be particularly helpful when assessing hip, knee, or ankle pain.<sup>1</sup> Moreover, an arthritic patient often has a limited range of motion on active and passive evaluation, but a patient with periarticular involvement or only mild noninflammatory articular disease often experiences pain on active range of motion with a relatively normal passive range of motion. Patients who have gout, an infected joint, or other severe inflammatory joint disease are readily identified because they resist any movement during examination.

### Inflammatory vs Noninflammatory Joint Disease

Inflammatory or noninflammatory joint disease can affect a single joint (monoarticular), two or three joints (oligoarticular), or four or more joints (polyarticular). It can also be classified as symmetric or asymmetric and primarily axial or peripheral. It is crucial to identify historical and clinical evidence of inflammation in order to proceed down the proper diagnostic pathways and to determine prognosis and treatment. For example, a patient with polyarthralgia will have a differential diagnosis list that is very different to a patient with oligo- or polyarticular inflammation. Poor conditioning, fibromyalgia, diabetes, hypothyroidism, Parkinson's disease, and depression are potential causes of diffuse pain from polyarthralgias, but none of these are due to inflammatory joint disease.

Characteristic features of inflammatory joint disease include prolonged morning stiffness after awakening and stiffening of joints later in the day after periods of inactivity (called gelling). Patients may experience relief of joint pain and stiffness reported after activity, but this is very different from the noninflammatory syndrome of osteoarthritis, in which the pain is made worse by activity. Other physical and historical findings of inflammatory joint pain include warmth, swelling, redness, tenderness, pain, and loss of function. Table 1 summarizes the key differences between inflammatory and noninflammatory joint pain.

Sign or Symptom	Inflammatory Joint Pain	Noninflammatory Joint Pain
Warmth	Yes	No
Swelling	Yes	No
Morning stiffness	Present for ≥15-20 minutes	None or present for <15 minutes
Gelling	Yes	None or present for <15 minutes
Pain	During activity and rest; stiffness often relieved by activity	During activity but improves with rest

### Pattern of Joint Disease

An evaluation of the pattern of involved joints includes:

- Number of joints
- Large joints (eg, hip, shoulder, knee) versus small joints (eg, wrist, ankle, metacarpophalangeal [MCP] or distal interphalangeal [DIP] joints)
- Symmetrical versus asymmetrical
- Axial versus peripheral
- Primarily upper extremity versus primarily lower extremity versus no clear pattern

A description of joint involvement alone will help to identify the most likely diagnoses. For example, an acute monoarthritis is suggestive of a possible infection, crystal-induced arthritis, or trauma, whereas bilaterally symmetric polyarthritis is more suggestive of the inflammatory joint diseases, such as RA, systemic lupus erythematosus (SLE), PsA, or viral arthritis. Differences in the pattern of joint involvement also facilitate diagnosis. For example, RA of the hand usually involves the proximal interphalangeal (PIP) and MCP joints but not the DIP joints, whereas PsA often affects the DIP joints. Symmetric arthritis is typical of RA, SLE, other connective tissue diseases, and some viral arthritides, whereas asymmetric arthritis is more characteristic of PsA and gout. Axial inflammation of sacroiliac joints or the spine is suggestive of AS or other related spondyloarthropathies. An oligoarthritis primarily affecting the lower extremity is suggestive of reactive arthritis.

### Presence of Extra-Articular Features.

While the diagnosis of inflammatory joint disease may be obscured by the presence of only musculoskeletal features at the time of initial evaluation, an accurate diagnosis may depend on the presence or absence of extra-articular features. A history of extra-articular features should be obtained to appreciate the sequential development of a multisystem process, and patients may only recall these features upon direct questioning. Syndromes often evolve, and a patient who may initially appear to have RA can develop the characteristic features of SLE or PsA. However, the presence of these features alone is not diagnostic. Table 2 summarizes selected extra-articular features by their associated disorders.

Feature	Disorder
Constitutional symptoms (eg, fatigue, malaise, weight loss)	AS
	PsA
	RA
	SLE
Ocular symptoms	AS (anterior uveitis)
	PsA (anterior uveitis)
	RA (scleritis, Sjögren or sicca complaints)
	ReA (anterior uveitis)
	SLE (conjunctivitis, cytooid bodies, Sjögren or sicca complaints)
Skin lesions	AS (keratoderma blenorrhagicum)
	Gout (tophi)
	PsA (psoriatic plaques)
	RA (nodules, vasculitis)
	Septic arthritis (embolic phenomena, Osler nodes, pustules)
	ReA (skin rash, genital lesions)
SLE (photosensitivity, malar rash, discoid skin lesions)	
Nail changes	PsA
	ReA

ReA = reactive arthritis.

### Laboratory Evaluation

Laboratory tests commonly used in patients with inflammatory joint disease include erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), rheumatoid factor (RF), and antinuclear antibody (ANA).<sup>3</sup> Elevated ESR or CRP levels are nonspecific indicators of an inflammatory process, but they do not identify a specific diagnosis. CRP increases more rapidly than ESR after the onset of inflammation and is less susceptible to local laboratory conditions. However, normal ESR or CRP levels do not rule out an inflammatory process. In fact, some patients with inflammatory disease, such as polymyalgia rheumatica, may have normal ESR and CRP levels.

RF, which is present in approximately 80% of patients with RA, is often used to confirm the suspicion of RA, but only 30% of patients with RA are positive for RF early in their disease.<sup>4</sup> Many other diseases, rheumatologic and otherwise, may be associated with a positive RF. Normal aging is associated with an increasing prevalence of RF positivity, and more than 30% of normal people in their eighth decade of life are RF positive. A more recent test for RA is the anticyclic citrullinated protein (CCP) assay, but the usefulness of this test in general practice is not yet fully established.

ANA is a sensitive marker for SLE, which is present in more than 95% of patients.<sup>4</sup> However, ANA should be used only when the clinical features of a case suggest SLE or other connective tissue disorder. It should never be used as a screening test because patients with other rheumatologic and non-rheumatologic diseases can be ANA positive but not have SLE. In addition, some healthy people without SLE, especially close relatives of SLE patients can be ANA positive.

The key takeaway is that laboratory tests can, in some cases, be helpful in confirming the presence of inflammatory disease. However, they are not diagnostic and are never useful as screening tools in the absence of strongly suggestive clinical evidence of disease. Moreover, typical serologic findings of certain inflammatory joint diseases may not be evident in the first month or two. A positive serologic test can also be misleading since the frequency of abnormal test results, such as ESR, RF, and ANA, increases with age even in healthy patients.<sup>5</sup>

### Synovial Fluid Analysis

Although a history and physical examination will differentiate between inflammatory and noninflammatory joint disease, analysis of the synovial fluid can also be used. Normal fluid appears transparent or slightly yellow or straw-colored, whereas inflammatory fluids are yellow, may have grossly obvious debris, and may be translucent to opaque. A popular technique of evaluating the synovial fluid for inflammation is the “newspaper test—newsprint print cannot be read through the

vial of inflammatory fluid. Table 3 summarizes the differences between normal, noninflammatory, and inflammatory synovial fluid. The best use of synovial fluid analysis is to identify the presence of crystals (eg, monosodium urate crystals in patients with gout and calcium pyrophosphate dihydrate crystals for pseudogout) and to identify if an infection is present by Gram stain and culture of the fluid.

### Imaging

Radiologic evidence of inflammatory joint disease is not typically present early in the disease process, and x-rays show no change or nonspecific changes, such as soft tissue swelling. The classic radiographic features of RA or PsA appear only after months of disease (see Table 4). In contrast, lupus arthritis does not cause erosions, osteopenia, or irreversible subluxations.

<b>Disease</b>	<b>Radiologic Features</b>
Rheumatoid arthritis	Periarticular osteopenia Joint space narrowing Marginal erosions
Psoriatic arthritis	Bony erosions at the cartilaginous edge “Pencil-in-cup” deformities
Osteoarthritis	Osteophytes Joint space narrowing Bony cysts and bony sclerosis

## Referring Your Patient to a Rheumatologist

### Why You Should Refer Patients with Inflammatory Joint Disease

Most studies comparing the long-term effects of early versus delayed treatment have been conducted in patients with RA. These clinical studies indicate that early use of disease-modifying antirheumatic drugs (DMARDs) in patients with RA is associated with reduced disease activity and reduced or slowed

	<b>Normal</b>	<b>Noninflammatory</b>	<b>Inflammatory</b>
<b>Gross examination</b>			
Viscosity	High	High	Low
Color	Colorless	Clear/yellow	Yellow/white
Clarity	Transparent	Transparent	Translucent/opaque
<b>Cell count</b>			
WBC count	<200/ $\mu$ g	200-2,000/ $\mu$ g	2,000-100,000/ $\mu$ g
PMN, %	<25	<25	>50

WBC = white blood cell; PMN = polymorphonucleocytes.

radiographic disease progression after several years of treatment.<sup>8-10</sup> There is evidence that nonrheumatologists delay the initial treatment of RA, and pharmacologic therapy is started more frequently if patients with RA have at least some contact with a rheumatologist.<sup>11</sup> In fact, patients managed by a rheumatologist experience better long-term outcomes.<sup>12,13</sup> The same principles of early diagnosis and treatment can be applied to other forms of inflammatory joint disease. Thus, most patients should not be initially managed by the primary care physician (PCP) *alone* because of their lack of clinical expertise with this patient population and limited experience of using the new biologic treatment options.

### Whom and When to Refer

Patients with a presumed or established diagnosis of inflammatory joint disease should be referred to a local rheumatologist. This referral can be based on the clinical suspicion of inflammatory joint disease and not a definitive diagnosis. More importantly, absence of serologic markers should not preclude referral to a rheumatologist if laboratory tests are performed.

The time to referral is dependent on two variables: (1) the time between onset of symptoms and consultation with a PCP and (2) the time between PCP consultation and referral to the rheumatologist.<sup>14</sup> While the PCP cannot control the first variable, they can unduly influence the second. Any patient with inflammatory joint disease should be referred to a rheumatologist as early as possible because initiation of therapy should not be delayed beyond several months. Although PCPs more frequently recognize the need for early referral and the time to referral is getting progressively shorter, less than half of the patients who are referred have symptoms less than 3 months.<sup>14</sup>

### How to Refer

It is important to identify and cultivate a relationship with an individual rheumatologist or rheumatology group in your local area. They may be identified through local networks and hospitals or through the Arthritis Foundation (contact information is at the end of this article). The PCP should contact the rheumatologist directly and maintain an open and active bidirectional dialogue. Although the rheumatologist is motivated to see the patient early in the disease process, it is the PCP's responsibility to indicate a certain degree of urgency. This makes the rheumatologist more likely to respond to the PCP's request relatively quickly, particularly if there is a certain level of clinical suspicion for an inflammatory joint disease.

### Role of the Rheumatologist

The rheumatologist's primary objective is to make or confirm a diagnosis, determine the prognosis, and provide a treatment

## Sample Case Presentation

"Doctor Smith, I have a 38-year-old woman with bilaterally symmetric inflammatory joint disease affecting the small joints of her hands and feet with 1 hour of morning stiffness and the presence of gelling. She has no psoriatic or other skin lesions. ESR is 57 mm/hr and CRP is 8 mg/dL. CBC shows mild thrombocytosis and a mild anemia with normal indices. She is feeling slightly better after 2 weeks of ibuprofen 600 mg PO QID, but it does not appear to be entirely relieving her discomfort. I feel she may need additional evaluation and treatment."

plan. The importance of a thorough patient history and physical examination by a specialist cannot be overemphasized in a group of diseases where clinical presentation is more important than the presence or absence of serologic markers. In general, a rheumatologist will initiate more aggressive treatment early in the disease, thus increasing the chance for improved long-term outcomes. The rheumatologist does not want to "steal" the patient away from the PCP and has no interest in interfering with the long-term relationship between the patient and health care provider. In fact, this relationship can help solidify patient acceptance of the treatment plan that the rheumatologist and PCP develop. Thus, the PCP and rheumatologist need to combine their efforts and work together during follow-up and maintenance. It is particularly important for the PCP to learn about the potential toxicities of the drugs recommended by the rheumatologist, as it is very likely that any complaints will be brought to the attention of the PCP.

## Conclusion

In general, most patients should not be initially managed solely by the PCP for their inflammatory joint disease because of the PCP's unfamiliarity with the complexities of disease management. The PCP should prioritize getting the patient to a rheumatologist early in the disease process and then co-manage the patient during follow-up. Once the rheumatologic syndrome is under control, the PCP has much more exposure to the patient and should therefore be the first line of defense. Consequently, early referral for diagnosis and treatment will improve patient outcome and long-term quality of life.

## Arthritis Foundation Contact Information

<b>Address:</b>	<b>P.O. Box 7669 Atlanta, GA 30357-0669</b>
<b>Phone:</b>	<b>1-800-283-7800</b>
<b>Web site:</b>	<b><a href="http://www.arthritis.org/">http://www.arthritis.org/</a></b>

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## Post-Test

1. Hallmarks of inflammatory joint disease that allow differentiation from noninflammatory joint disease include all of the following **except**:
  - A. Warmth of the joint
  - B. Swelling of the joint
  - C. Morning stiffness of more than 1 hour
  - D. Pain and stiffness made worse by activity
  - E. Return of stiffness after prolonged periods of rest
2. The syndrome least likely to cause a bilaterally symmetric distribution of inflammatory joint disease is:
  - A. Lupus
  - B. Rheumatoid arthritis
  - C. Gout
  - D. Viral arthritis
  - E. Psoriatic arthritis
3. Constitutional complaints are common in all of the following syndromes **except**:
  - A. Lupus
  - B. Osteoarthritis
  - C. Rheumatoid arthritis
  - D. Ankylosing spondylitis
  - E. Psoriatic arthritis
4. Radiographic abnormalities in psoriatic arthritis are commonly seen early in the course of disease, in contrast with the delayed radiographic changes seen in rheumatoid arthritis.
  - A. True
  - B. False
5. Early referral to a rheumatologist has been shown to correlate with improved long-term outcomes in a number of rheumatologic disorders.
  - A. True
  - B. False

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04-SC-43-W

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### Upon completion of this activity, are you able to:

Identify and differentiate inflammatory joint disease from noninflammatory disease?

Yes

No

Describe how to identify extra-articular features that help diagnose inflammatory joint disease syndromes?

Yes

No

List the laboratory tests that help to confirm the presence of inflammatory disease and assist in making a specific diagnosis?

Yes

No

Identify the importance of early referral to the rheumatologist?

Yes

No

### Please rate the monograph using the scale below and check the appropriate box.

Value of topic

Poor

Fair

Satisfactory

Good

Excellent

Rate the overall clinical relevance of this monograph to your practice needs

Poor

Fair

Satisfactory

Good

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Did the program meet your expectations?

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Was the content of this monograph free of commercial bias?

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No

If no, why not? \_\_\_\_\_

What one new thing did you learn today? \_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_

What change will you make to your practice performance as a result of attending this program? \_\_\_\_\_  
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\_\_\_\_\_

What recommendations do you suggest to improve this program? \_\_\_\_\_  
\_\_\_\_\_  
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What topics would you like to see in future arthritis presentations? \_\_\_\_\_  
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Record your answers below by circling the appropriate letter.

- 1. A B C D
- 2. A B C D
- 3. A B C D
- 4. A B
- 5. A B