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A Publication for Rheumatology Nurse Practitioners and Physician Assistants

Spondyloarthropathy: Recognizing the Undiagnosed Patient

Martin J. Bergman, MD, Senior Editor

The spondyloarthropathies (SpAs) are a group of interrelated, chronic inflammatory arthritides that includes ankylosing spondylitis (AS), reactive arthritis (ReA), psoriatic arthritis (PsA), enteropathic arthritis (arthropathy of inflammatory bowel disease [IBD]), and undifferentiated SpAs.¹⁻³ Although they share certain genetic predisposing factors and clinical features, the SpAs are heterogeneous disorders. Diagnosis is based on clinical findings; diagnostic criteria developed for research generally are not useful in clinical practice.² Because of the recent development of effective biologic therapies, early diagnosis of SpA is crucial.⁴ Delays in diagnosis, however, are common. In patients with AS, for example, the average diagnosis delay is 9 to 11 years after onset of symptoms—long enough for permanent damage to develop.⁵

This issue of PCE Updates in Rheumatology focuses on early recognition of SpAs and strategies for differentiating among AS, PsA, ReA, and IBD-associated or undifferentiated SpA. Rick Pope, MPAS, PA-C, presents a case study of a patient with SpA with clinical commentary (page 6).

Needs Assessment

The spondyloarthropathies (SpAs) are a group of related inflammatory arthritides, including ankylosing spondylitis (AS), reactive arthritis, psoriatic arthritis (PsA), arthropathy of inflammatory bowel disease (IBD), and undifferentiated SpAs. As a group the SpAs are among the most common rheumatic diseases, with a prevalence ranging from 0.5% to 1.9%; the most common SpA subgroups are AS and undifferentiated SpA.¹ Diagnosis is largely based on clinical findings; however, differentiating among the SpAs, particularly in early stages, may be difficult because of overlapping features.² Because of the recent development of effective biologic therapies, early diagnosis of SpA is crucial. Delays in diagnosis are common. In patients with AS, for example, the average diagnosis delay is 9 to 11 years, long enough for permanent damage to develop.³

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Target Audience

Nurse practitioners (NPs), physician assistants (PAs), and physicians in the practice of rheumatology.

Learning Objectives

After completing this activity, participants should be better able to:

1. Identify early signs of SpAs in patients with rheumatologic complaints.
2. Differentiate among AS, PsA, ReA, and IBD-associated or undifferentiated SpA, based on their characteristic features.
3. Use biologic agents in treatment plans for specific SpAs.

(continued on page 2)

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Perspectives From Rick Pope, MPAS, PA-C

What challenges do physician assistants face in diagnosing SpAs?

The incidence of SpAs in this country may surpass that of rheumatoid arthritis (RA),⁶⁻⁸ and now that we have effective therapies, it is incumbent on us to think about this group of diseases. If we search for a spondylitis diagnosis, we will find it in some unlikely patients. The case presented on page 6 was highly unusual in that the gender and age of the patient did not fit the typical pattern expected for this group of diseases. The diagnosis in this case would have been easy to miss.

What approach do you use in selecting treatment for patients with spondylitis?

In the past, I have used inflammatory markers as a guide to identifying patients who require aggressive treatment. However, many patients with spondylitis



have a normal erythrocyte sedimentation rate (ESR) and/or C-reactive protein (CRP) level. Even in these patients, back stiffness and peripheral joint symptoms improve with tumor necrosis factor (TNF) inhibitor therapy. Many patients also report improvements in their energy level when TNF inhibitors are used. In my experience, the use of methotrexate has been less than satisfactory in patients with spondylitis.

What are the take-home messages for diagnosing spondylitis?

Remember that classic male presentation in the 24- to 26-year-old age group does not fit all cases of spondylitis. As noted in the case study (page 6), women—even elderly women—can present with this disease. Until recently, spondylitis has received less attention than RA. In 2009, however, with improved treatments for spondylitis and the knowledge that there are many patients with undiagnosed spondylitis in the population, it becomes incumbent on us to consider this diagnosis. I am reminded of the medical axiom, “If you think of the diagnosis, you will find it.”

Epidemiology and Characteristic Features

SpAs are among the most common rheumatic diseases, with a prevalence ranging from 0.5% to 1.9%.⁹ Of the SpA disorders, AS and undifferentiated SpA are the most common. SpAs have a male preponderance, although both genders are affected. The onset of SpAs occurs at all ages but usually begins in late teens and early 20s. In pediatric patients, ~20% of all chronic arthropathies are probably due to SpAs.¹

The SpAs are linked together by a common gene and by a common pathology.^{1,2} Although the SpAs are strongly associated with HLA-B27 gene, this association varies among different SpAs (Table 1) and among different ethnic groups, and it may be absent in some patients.¹ The basic pathologic lesion in SpAs is enthesitis, which occurs most commonly in the axial skeleton and the

lower extremities (Achilles tendon, plantar fascia, patella tendon, and metatarsal heads).^{1,2} Other characteristic features of the SpAs include increased familial incidence; associations with psoriasis, other mucocutaneous lesions, or chronic IBD; variable peripheral arthritis, and dactylitis; radiographic sacroiliitis with or without spondylitis; ocular inflammation; and occasional aortitis or heart block.¹ The etiology of SpA is unknown but exposure to an infectious agent or an antigen in a HLA-B27–positive or otherwise susceptible patient may be a trigger.²

Diagnostic Challenges

Classification and diagnostic criteria for SpAs are limited.^{1,4,10,11} The most widely used classification criteria for SpAs were developed in 1991 by the European Spondylarthropathy Study Group (ESSG).¹⁰ According to these criteria, presence of inflammatory

Table 1. Characteristic Features of Common SpAs^a

Feature	AS	PsA	Enteropathic Arthritis	ReA	Undifferentiated SpA
Age at onset	Late teens-early adulthood	35-45 years	Any age	Late teens-early adulthood	NR
HLA-B27 association	90%-95%	40%	30%	80%	NR
Male:female ratio	3:1	1:1	1:1	5:1	NR
Dactylitis	Uncommon	Common	Uncommon	Common	May be present
Enthesitis	Common	Very common	Occasional	Common	May be present (especially at the heel)
Peripheral arthritis Frequency Distribution	Occasional Asymmetric, lower limbs	Common Asymmetric, any joint	Common Asymmetric, lower limbs	Common Asymmetric, lower limbs	Occasional Lower extremities
Sacroiliitis Frequency Distribution	100% Symmetric	40% Asymmetric	20% Symmetric	40%-60% Asymmetric	NR
Syndesmophytes	Delicate, marginal	Bulky, nonmarginal	Delicate, marginal	Bulky, nonmarginal	NR
Uveitis	Acute anterior uveitis, usually unilateral and recurrent	Chronic uveitis, can be bilateral	Chronic uveitis, usually bilateral	Acute anterior uveitis, conjunctivitis	Acute anterior uveitis (occasional)
Skin lesions	None	Psoriasis	Erythema nodosum, pyoderma gangrenosum	Circinate balanitis, keratoderma, blennorrhagicum	NR
Nail changes	None	Pitting, onycholysis	Clubbing	Onycholysis	NR
Cardiac manifestations	Aortic regurgitation, conduction defects	Aortic regurgitation, conduction defects	Aortic regurgitation	Aortic regurgitation, conduction defects (rare)	Aortic regurgitation, conduction defects (occasional)

NR = not reported.

^aThis categorization of the SpAs is evolving. Clinicians are beginning to look at SpAs as axial or peripheral disease with variable extra-articular manifestations.³ Khan M¹; Kataria RK et al²; van der Linden SM et al.¹⁶

spinal pain or synovitis (asymmetric or predominantly in the lower limbs), together with at least 1 of the following—positive family history, psoriasis, IBD, urethritis, or acute diarrhea, alternating buttock pain, enthesopathy, or sacroiliitis as determined from radiography of the pelvic region—are required for a classification of SpA.¹⁰ However, a study that evaluated the performance of the ESSG criteria as diagnostic criteria in clinical practice found that only 47% of patients with possible SpA who met the ESSG criteria at entry into the study were judged by their rheumatologists to have SpA after 5 years of follow-up.¹²

As there are no specific diagnostic tests for SpAs, diagnosis is based primarily on patient history, physical examination, and the experience of

the clinician.^{2,9} Laboratory findings of rheumatoid factor negativity, elevation of inflammatory markers (ESR or CRP), and presence of anemia of chronic disease may support the diagnosis of SpA but are nonspecific.² Neutrophils abound in synovial fluid aspirates of patients with white blood cell counts exceeding 2000 cells/mL, which is indicative of inflammation.² This finding also is nonspecific. HLA-B27 testing is not clinically helpful because SpAs can occur in the absence of the allele.^{1,2} The genetic phenotype occurs in 8% of healthy white persons, but 90% of them will never develop SpAs. Radiographic analysis of the pelvis and lumbar spine may show evidence of sacroiliitis or spondylitis

after prolonged disease; radiographs are not useful in early disease.⁴ Recent reports suggest that magnetic resonance imaging might be a useful tool for early diagnosis.^{13,14}

Keys to Early Recognition

With the advent of effective biologic therapies for SpAs (see *Fast Facts: Anti-TNF- α Therapies for Spondyloarthropathies and Associated IBDs*, page 5), early recognition and differentiation among SpAs are critical to improving clinical outcomes. In a study of patients with active AS, patients with a short disease duration and good functional status were more likely to respond to anti-TNF- α agents than patients with longstanding disease and impaired function.¹⁵

Particularly in early stages, differentiating among the SpAs can be difficult because of overlapping features (Table 1).^{1,2,16} The clinician has to rely on thorough evaluation of clinical symptoms and presentation patterns to make the correct diagnosis.

Ankylosing Spondylitis

AS, characterized by spinal stiffness and sacroiliitis, occurs in 0.1% to 0.2% of the US general population.² Although radiographic evidence of bilateral sacroiliitis usually is needed for a definite diagnosis of AS, radiographic sacroiliitis is neither an early nor an obligate manifestation of AS.¹⁶ The presence of inflammatory low back pain and stiffness and a positive family history for AS are 2 important features that may help in early diagnosis.^{16,17} Inflammatory back pain may be suspected when low back pain begins in a patient <45 years old (as new onset AS is rare after this age) and is associated with any 2 of the following features: morning stiffness lasting for at least 30 minutes, pain that improves with exercise but not with rest, pain that awakens the patient during the second half of the night only, and pain that alternates between buttocks.¹⁷ A positive family history increases the probability of AS from 0.1% (prevalence of AS in the general population) to ~10% if the patient has a first-degree relative with AS; the probability increases to ~50% if the relative has inflammatory low back pain.¹¹ Additionally, the presence of inflammatory back pain together with 3 other SpA features, including family history, increases the probability of axial SpA (which includes AS and undifferentiated SpA) to ~90%.⁹ Using inflammatory low back pain, family history, and presence of SpA features, clinicians can diagnose AS with a high degree of confidence at an early stage in patients presenting with low back pain without evidence of radiographic sacroiliitis.⁹

Patients with late-stage AS may present with axial arthritis involving

the cervical spine and limited spinal mobility. In these patients, the Schober's test may be useful for measuring spinal mobility, but it is nonspecific for diagnosing AS.² The hips and shoulders (often early in the disease course) and peripheral joints (usually later in the disease course) also are affected by arthritis.

Extra-articular manifestations, excluding dactylitis, are common in patients with AS.² Anterior uveitis, the most common extra-articular feature, occurs in 25% to 30% of patients with AS.¹⁸ The uveitis in AS usually is acute, unilateral, and recurrent. Both aortitis and cardiac conduction abnormalities, particularly heart blocks, are extra-articular manifestations that also should be considered in this group of patients.

Enteropathic Arthritis

Enteropathic arthritis is arthropathy associated with IBD, Crohn's disease, or ulcerative colitis. Up to 20% of patients with IBD have arthropathy; but, arthritic symptoms may manifest before clinical bowel disease.²

Arthritic symptoms usually have a sudden onset and typically affect the lower extremities (peripheral arthritis) asymmetrically. The arthritis has a migratory pattern and generally is self-limiting and nondeforming. Recurrence is common, and 10% of patients develop chronic arthritis. Axial involvement occurs in ~20% to 25% of patients and could resemble that in AS (bilateral sacroiliitis).^{1,2} Sacroiliitis also may occur asymmetrically and may not be associated with back pain.¹⁹ Peripheral arthritic symptoms usually coincide with IBD activity, while axial disease is independent of IBD activity. Uveitis and skin lesions are chronic in IBD-associated arthritis and the former usually occurs bilaterally.²

Psoriatic Arthritis

Between 0.2% and 0.4% of the US general population is afflicted with PsA, and ~20% of patients with psoriasis have PsA.² Although psoriasis

precedes arthritic symptoms by about 10 years in most cases, arthritis occurs without skin symptoms in 15% to 30% of cases.²⁰ There are 5 subtypes of PsA, which are based on symptoms at disease onset: oligoarticular (≤ 4 joints affected); polyarticular (≥ 5 joints affected); a pattern with predominant distal interphalangeal (DIP) joint involvement; arthritis mutilans; and psoriatic spondylitis.² The distinction among these subtypes becomes less clear with disease progression and may not be useful in the diagnosis of PsA. A more recent classification of PsA, based on the discrete clinical presentations of PsA—skin and nail disease, peripheral arthritis, axial disease, dactylitis, and enthesitis—might be more useful from a diagnostic and treatment perspective.²¹

Arthritis in PsA usually is asymmetric and affects the distal joints; ~40% of patients have asymmetric sacroiliitis.² The severity of arthritic symptoms may range from mild remitting arthritis to erosive disabling disease (arthritis mutilans)²; the latter occurs in ~20% of patients.²⁰ Radiographic changes associated with erosive disease could include the typical pencil-in-cup deformity of DIP joints. Nail lesions, including pitting and onycholysis, are common in PsA occurring in >80% of patients.^{2,20} Psoriatic lesions typically occur on the knees and elbows, but other sites such as the scalp, ears, anal cleft, perineum, or umbilicus, may also be involved.^{1,2}

Reactive Arthritis

ReA, an aseptic peripheral arthritis triggered by an infectious agent located outside the joint, has a prevalence of ~0.1%.² Arthritic symptoms appear about 4 weeks after an infection, usually a genitourinary infection with *Chlamydia trachomatis* or enteritis due to gram-negative enterobacteria, such as *Shigella*, *Salmonella*, *Yersinia*, or *Campylobacter* species.^{1,2} ReA is characterized by acute, asymmetric,

Fast Facts: Anti-TNF- α Therapies for Spondyloarthropathies and Associated IBDs

TNF- α plays a central role in the pathogenesis of AS and PsA. TNF- α gene expression is elevated in synovial tissues of sacroiliac joints, and TNF- α levels are elevated in psoriatic joint fluid.²² Increased expression of TNF- α has been found in the intestinal mucosa of patients with Crohn's disease, indicating a role for TNF- α in the pathogenesis of IBD.²³ In controlled trials of AS, the use of anti-TNF- α agents etanercept, infliximab, and adalimumab improved function and spinal and peripheral joint pain.²⁴⁻²⁷ These anti-TNF- α agents also are effective in controlling psoriatic joint inflammation and inhibiting radiographic progression; however, adalimumab and infliximab are more effective than etanercept in the treatment of psoriasis.²² Adalimumab, infliximab, and the recently approved anti-TNF- α agent certolizumab have been shown to be effective in reducing the signs and symptoms associated with Crohn's disease.

Adalimumab and infliximab are also effective in inducing and maintaining clinical remission in Crohn's disease.²⁸⁻³⁰ Infliximab also is effective in reducing the signs and symptoms of ulcerative colitis and inducing and maintaining clinical remission and mucosal healing.²⁹ Etanercept has not been approved for IBD.³¹

Prior to the introduction of biologics, therapy with nonsteroidal anti-inflammatory drugs (NSAIDs) and nonbiologic disease-modifying antirheumatic drugs, such as methotrexate, were the pharmacologic options for SpAs. Although these therapies can be used in patients with less severe disease, they are often ineffective in patients with severe peripheral arthropathy and axial involvement.³² Additionally, NSAID use in patients with enteropathic arthritis is associated with the risk of exacerbating underlying IBD.¹⁹ The anti-TNF- α agents now offer an alternative, more effective treatment option for SpAs.

SpA		
Anti-TNF- α Agent	Disease	Dosage
Adalimumab	AS (active)	40 mg SC every other week
	PsA (active)	40 mg SC every other week
Etanercept	AS (active)	50 mg SC/week
	PsA (active)	50 mg SC/week
Infliximab	AS (active)	5 mg/kg IV, followed by additional similar doses at 2 and 6 weeks after first infusion, and then every 6 weeks thereafter
	PsA (active)	5 mg/kg IV, followed by additional similar doses at 2 and 6 weeks after first infusion, and then every 8 weeks thereafter
IBD		
Adalimumab	Crohn's disease (moderately to severely active) ^a	<i>Initial dose:</i> (Day 1) 160 mg SC (4 40-mg injections in 1 day or 2 40-mg injections per day for 2 consecutive days; followed by 80 mg SC 2 weeks later (Day 15) <i>Maintenance dose:</i> 2 weeks later (Day 29) 40 mg SC every other week
Certolizumab	Crohn's disease (moderately to severely active)	<i>Initial dose:</i> 400 mg SC initially and at Weeks 2 and 4 <i>Maintenance dose:</i> 400 mg SC every 4 weeks
Infliximab	Crohn's disease (moderately to severely active or fistulizing)	5 mg/kg IV at 0, 2, and 6 weeks; followed by maintenance therapy with 5 mg/kg IV every 8 weeks thereafter ^b
	Ulcerative colitis (moderately to severely active)	5 mg/kg IV at 0, 2, and 6 weeks; followed by maintenance therapy with 5 mg/kg IV every 8 weeks thereafter

^aAdalimumab is also indicated for reducing signs and symptoms and inducing clinical remission in patients with moderately to severely active disease who have lost response to or are intolerant to infliximab.

^bFor adults who respond and then lose their response, treatment with 10 mg/kg IV may be considered; patients who do not respond by week 14 are unlikely to respond and treatment discontinuation should be considered.

SC = subcutaneous.

Data from prescribing information for adalimumab,²⁸ infliximab,²⁹ certolizumab,³⁰ and etanercept.³¹

and oligoarticular arthritis of the lower extremities that is associated with ≥ 1 extra-articular feature: ocular inflammation (conjunctivitis or uveitis), enthesitis (Achilles tendonitis and plantar fasciitis), dactylitis, mucocutaneous lesions, and urethritis. Cardiac manifestations are rare.^{1,2} Extra-articular manifestations are common in ReA and

important in establishing the diagnosis. Although the triad of arthritis, conjunctivitis, and urethritis can occur in ReA, most patients with ReA do not present with this triad.¹ For example, conjunctivitis occurs in $\leq 50\%$ of patients with ReA, but is not always associated with arthritic flares.² Enthesitis with periosteal reaction, asymmetric sacroiliitis, and

discontinuous spondylitis with bulky nonmarginal syndesmophytes are some characteristic radiographic features of ReA.² ReA usually is self-limiting with symptoms lasting 3 to 12 months. Recurrence of arthritis, however, is common, affecting $\leq 50\%$ of patients. Chronic arthritis or sacroiliitis develops in 15% to 30% of patients.²

Case Study: Evaluation of an 82-Year-Old Woman With Knee Effusions and Back Stiffness

Rick Pope, MPAS, PA-C

Presentation

An 82-year-old white woman presented with recurrent right knee effusions. She had no complaints of instability in either knee. The effusions, which had started in the spring of 2008, were aspirated twice by an orthopedist, but she was not called back for a follow-up visit. When she developed a third knee effusion in <6 months, her primary care clinician ordered a magnetic resonance imaging (MRI) scan. The MRI showed a moderate joint effusion; osteoarthritis with mild joint space narrowing in the medial compartment; an oblique tear of the posterior horn of the medial meniscus; and several complex tears throughout the rest of the meniscus. She was referred for a rheumatology consultation in mid-August.

History

The patient had been cleaning out her deceased sister's house and going up and down stairs frequently when her knees began to swell, but she had not related this increased activity to the onset of her knee effusion. She reported no other joint swelling or pain. However, for the past 10 years, she has been plagued with bilateral buttock and back stiffness, which lasts up to 2 hours. A physician once suggested that she might have spondylitis, but she did not pursue consultation. She underwent a left hip replacement for presumed osteoarthritis and has no known family history of arthritis.

She complained of chronic dry eyes and discomfort when exposed to sunlight but reported no hot, red, swollen painful eye conditions; fevers; or skin rashes. She had no known history of a tick bite or a recent flu-like illness. She has gastroesophageal reflux disease (GERD), but no history of other gastrointestinal (GI) problems. She had no genitourinary or cardiac complaints, and her medical history was otherwise unremarkable.

Physical and Laboratory Findings

- Right knee: slightly warm, with a moderate to large effusion; slight degree of flexion contracture secondary to the effusion
- Back: limited flexion during toe touching
- Right knee aspirate: 40 mL clear yellow fluid; negative for crystals; low cell counts with an excess of granulocytes; Gram staining and cultures, negative
- Lyme disease antibody titer: negative
- CRP: normal
- Complete blood cell count and chemistry profile: within normal limits

Radiographic Findings

Radiographs of the sacroiliac joint (SIJ) and lumbosacral (L-S) spine were ordered.

- SIJ: ankylosis, complete on the left and nearly complete on the right; only nonfused area, at the superior margin on the right

- L-S spine: hyperlordosis on the lateral view with narrowing of the disc space at T12, L1; generalized osteopenia, diffuse osteophytosis, no clear evidence of syndesmophytes

Diagnosis

The patient's medical history and physical, laboratory, and radiographic findings are consistent with a diagnosis of undifferentiated spondylitis and an unrelated osteoarthritic knee with internal derangement.

Clinical Commentary

The noninflammatory nature of the patient's right knee effusion suggested osteoarthritis, and the MRI revealed internal derangement. However, her history and radiographic findings fit a pattern of spondylitis, and she likely has had symptoms of this disease for more than 10 years. She has the characteristic back and buttock stiffness, lasting 1 to 2 hours and improving with movement. Radiographs of the SIJ showed bilateral sacroiliitis. Radiographs of the lumbar spine did not reveal syndesmophytes (calcification of the longitudinal ligaments) or squaring of the anterior portion of the vertebral bodies, suggesting that her disease had not progressed upward from the SIJs. The question also arises as to whether her need for a hip replacement was caused by inflammation or merely wear and tear in the joint. If inflammation had been present, the radiographs likely would have shown symmetric narrowing with secondary osteoarthritis, in contrast to the asymmetric narrowing seen in osteoarthritis. Spondylitis often affects the hip and shoulder joints, in addition to the spine.

Treatment Decision

Because the patient's history of GERD in addition to her age place her at high risk for GI bleeding, the decision was to prescribe treatment with celecoxib and a proton pump inhibitor (PPI). The option of a surgical referral for arthroscopic debridement of her knee was discussed with her, but she declined. Physical therapy also was prescribed to strengthen her quadriceps muscles, and she was instructed to avoid kneeling and excessive stair climbing. A 3-week follow-up visit was scheduled.

Patient Follow-up

After 3 weeks, she had no recurrence of the knee effusion, and her back stiffness had improved. She was tolerating treatment with celecoxib and a PPI. The decision was made to monitor the patient closely and make additional therapeutic decisions in a slow and thoughtful manner. Although she had 3 knee effusions within a short period, it was felt that a slower and less aggressive approach would be more prudent because of her age. The possibility of starting anti-TNF- α therapy if her symptoms progressed was discussed with her, and she was tested for tuberculosis and referred for a chest x-ray.

Undifferentiated Spondyloarthropathy

Patients with isolated clinical manifestations of SpA that are insufficient to be categorized into any of the well-defined SpAs are considered as having undifferentiated SpA. The presenting symptoms may include HLA-B27–associated oligoarthritis or polyarthritis, mostly of the lower extremities; dactylitis; enthesitis, especially at the heel (Achilles tendonitis and plantar fasciitis); inflammatory back pain; or unilateral or alternating buttock pain; and occasionally extra-articular manifestations (acute anterior uveitis or aortic incompetence and heart block).^{1,2} The extra-articular manifestations may be accompanied

by arthritis or may precede arthritic symptoms. Undifferentiated SpA is not associated with preceding bacterial infections, IBD, or psoriasis. Over time, undifferentiated SpA may progress to one of the other well-defined SpA disorders.

PCE Takeaways

- SpAs are a group of interrelated, chronic inflammatory arthropathies that are linked together by a common gene (HLA-B27) and a common pathology (enthesitis); however, they are distinct disorders and can occur in the absence of HLA-B27 positivity.
- SpAs share several common clinical features including

peripheral arthritis, axial involvement, and inflammation in periarticular structures.

- There are no specific diagnostic tests for SpA disorders; diagnosis is based primarily on patient history, physical examination, and the experience of the clinician.
- Early recognition of SpAs is critical for effective biologic therapies (anti-TNF- α agents) that are now available; however, early recognition and differentiation among the disorders is difficult because of overlapping clinical features.
- Inflammatory low back pain and stiffness and a positive family history for AS have been recognized as important features that may help in the early diagnosis of AS.

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About PCE Updates in Rheumatology...

Welcome to the fourth of 4 issues of *PCE Updates in Rheumatology*, Volume 2 developed for those NPs and PAs who serve patients in rheumatology practices throughout the country. "The demands for rheumatologic services are increasing exponentially with the aging population, while the number of physicians entering the rheumatology specialty is shrinking," according to the Society of Physician Assistants in Rheumatology (SPAR). Rheumatologists treating patients with rheumatoid diseases increasingly rely on specialist NPs and PAs to take an active management role in the frequent clinical contacts these patients require.

As healthcare professionals, NPs and PAs collectively provide a vital and increasing role in the diagnosis and management of acute and chronic illness. As clinicians, you spend more time with patients than most physicians, with your emphasis being patient disease state counseling and preventive care. Most importantly, NPs and PAs report that their roles have evolved from assisting physicians to treating and following their own patients. This increased role includes writing prescriptions,

monitoring patient progress, and seeing patients in your own examination rooms. As NPs and PAs, you are rapidly emerging as key providers of patient care. You practice with greater autonomy and prescribe more medication than ever before.

Approximately 286 NPs and 188 PAs see patients in rheumatology practices that provide ongoing care for patients with rheumatoid diseases. Therefore, you need to be thoroughly familiar with the latest therapeutic advances. This issue of *PCE Updates in Rheumatology* focuses on early recognition of spondyloarthropathies (SpA) and strategies for differentiating among ankylosing spondylitis, psoriatic arthritis, reactive arthritis, and inflammatory bowel disease-associated or undifferentiated SpA.

PCE Updates in Rheumatology are brought to you by Practicing Clinicians Exchange™, an educational initiative designed specifically for NPs and PAs that provides CE/CME-accredited live symposia and online home study activities. Visit us at www.practicingclinicians.com to learn more about this unique initiative specifically designed to meet your educational needs.