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EPIDEMIOLOGY (J GELFAND, SECTION EDITOR)

Epidemiology and Natural History of Psoriatic Arthritis: an Update *What Dermatologists Need to Know*

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Abstract Psoriatic arthritis (PsA) is a chronic inflammatory disease that can be associated with permanent joint damage and disability. Because patients may be unaware of the association of joint disease with psoriasis, dermatologists play an important role in identifying PsA. In this review, we discuss the natural history and key features of PsA, the epidemiology and hypothesized risk factors for disease, and screening tools that can be used in the dermatology clinic to aid in identifying which patients should be referred to a rheumatologist for further assessment.

Keywords Psoriatic arthritis · Epidemiology · Psoriasis · Incidence · Prevalence · Risk factors · Screening tools

Introduction

Psoriatic arthritis (PsA) is a chronic inflammatory disease that can be associated with permanent joint damage and disability. Identification and appropriate treatment can dramatically improve quality of life in patients with PsA [1, 2]. However, PsA often can be difficult to identify. In many patients with PsA, the symptoms of arthritis do not develop until years after the onset of cutaneous psoriasis. Dermatologists play a critical role in screening for PsA, because many patients are not aware of the link between joint disease and psoriasis. In this review, we address the epidemiology, natural history, and clinical characteristics of this disease, as well as available screening tools to provide dermatologists the information needed to identify patients with PsA.

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Characteristics of Disease and Natural History

Clinical Features

Psoriatic arthritis is an inflammatory arthritis involving the articular and periarticular structures and most often is associated with psoriasis. PsA was initially thought to be mild compared with rheumatoid arthritis (RA), but over time, the potential for aggressive, erosive, and deforming disease has been recognized [3]. PsA is a member of a larger group of inflammatory arthritides called the spondyloarthropathies. This group also includes ankylosing spondylitis, reactive arthritis, enteropathic arthritis (associated with inflammatory bowel disease), and undifferentiated spondyloarthropathy.

The key features of PsA are inflammatory arthritis, enthesitis, dactylitis, and spondylitis (Table 1). Patients may have any combination of these features. Inflammatory arthritis or synovitis is distinguished from noninflammatory arthritis by swelling, warmth, tenderness, and erythema, although not all inflamed joints will have all of these features. A distinguishing feature between PsA and other inflammatory arthritides is the involvement of the distal interphalangeal joints (DIPs). Dactylitis and enthesitis are both characteristic features of spondyloarthropathies (such as PsA) and are important to recognize. Dactylitis (also known as “sausage toe” or “sausage finger”)

Table 1 Key features of psoriatic arthritis

Feature	Inflammatory arthritis	Enthesitis	Dactylitis	Spondylitis
Definition	Also known as "synovitis:" inflammation of the synovium (the lining of the joints)	Inflammation of the enthesis, or site where ligaments, tendons, and/or fascia attach to bone	Inflammation of the entire digit, including the joints, adjacent soft tissues, and tendons	Inflammation of one or more vertebrae (intervertebral and costovertebral joints), or of the sacroiliac (SI) joints (sacroiliitis)
History	History of prolonged morning stiffness (>30-45 min); joint pain that improves with activity and worsens with rest; any joint can be affected, though DIP involvement raises suspicion for PsA over other types of inflammatory arthritis.	Pain at the heel, tibial tuberosity, iliac crest, and other tendon insertion sites [4–6]	Pain, swelling, and/or warmth of an entire digit	Back or hip pain that is worse in the mornings (with >1 hour of morning stiffness)
Exam features	Swelling, warmth, tenderness, and/or erythema of the joints (with a classic "boggy" sensation); not all inflamed joints will have all of the exam features	Tenderness, warmth, and/or swelling at enthesis, particularly the Achilles tendon in PsA [6]	Swelling, warmth, and/or erythema of the entire digit from its base (also known as "sausage toe" or sausage finger"); this is unlike synovitis, where inflammation is confined to the joints	Paravertebral and/or SI joint tenderness; abnormal Schober's test*

*Modified Schober's test: the examiner makes a mark at the level of L5 (fifth lumbar vertebrae). He then makes a second mark at 5 cm below the L5 mark and another at 10 cm above the L5 mark. The patient is asked to touch his toes. By doing so, the distance between the second and third marks should increase by at least 5 cm. If the distance increases less than 5 cm, it indicates reduced lumbar flexion due to possible vertebral inflammation [4, 5]

involves swelling of the entire digit from its base, unlike synovitis in which swelling is confined to the joints. This is a result of inflammation in the adjacent soft tissues and tendons. Enthesitis (or enthesopathy) refers to inflammation at the site where ligaments, tendons, and/or fascia attach to bone. The enthesis is composed of dense collagen and fibrocartilage and is adjacent to bursae and synovial tissue [7]. Enthesitis is usually associated with history of discomfort in the tendon or ligament, morning stiffness, and on examination, there is generally tenderness, warmth, and/or swelling. Enthesitis can occur at many sites, but the most common sites are the insertion of the Achilles tendon into the calcaneus and the plantar fascia. Spondylitis is defined as inflammation of one or more vertebrae (intervertebral and costovertebral joints) or of the sacroiliac (SI) joints (sacroiliitis). Patients with spondylitis present with back or gluteal pain that is typically worse in the mornings with greater than an hour of morning stiffness [4, 5].

Several patterns of joint involvement in PsA have been identified: monoarthritis, oligoarthritis (less than 5 joints affected in an asymmetric distribution), polyarthritis (similar to and at times indistinguishable from RA), distal interphalangeal joint (DIP) joint predominant arthritis, arthritis mutilans (characterized by deforming and destructive arthritis), and psoriatic spondylitis (including both sacroiliitis and spondylitis). Patients also may present with predominantly enthesial inflammation. Clinical presentation is quite variable with many patients having more than one feature. Moll and Wright initially described the most prevalent subgroup as oligoarthritis [8], but clinical patterns in recent studies have varied depending on the population observed [3, 9–15]. Among two studies observing patients with psoriasis in a dermatology clinic, one reported 59 % polyarthritis and 32 % oligoarthritis among those with PsA [16] and another reported 31 % polyarthritis and 31 % oligoarthritis [17]. The latter study excluded those with previously diagnosed PsA. Because patients with oligoarthritis may progress to polyarthritis, length of disease and therapies used among those with PsA may influence the prevalence of either subtype.

Defining PsA

The original diagnostic criteria of Moll and Wright were developed in 1973 [8] and include the following:

- inflammatory arthritis (peripheral arthritis and/or sacroiliitis or spondylitis)
- presence of psoriasis
- (usual) absence of serological tests for rheumatoid factor.

Although the Moll and Wright criteria continue to be widely used, they have been shown to discriminate poorly between PsA and RA [18]. Since that time, several classification criteria have been developed [19–24]. It is important to note that classification criteria are developed to provide uniform definitions for inclusion in research studies. While developed for use in research, these criteria also can be

helpful in guiding diagnosis in clinical practice [25]. Development of the ClASsification criteria for Psoriatic Arthritis (CASPAR) criteria in 2006 provided a promising framework for conducting clinical research in PsA, given its high sensitivity and specificity (each >0.9) in the rheumatology, early arthritis, and family practice settings (Table 2) [23, 25–29]. CASPAR criteria are currently the most widely accepted tool for defining PsA in clinical research studies and are commonly used to guide the diagnosis of PsA in clinical practice.

Onset of PsA Relative to Psoriasis Onset

Approximately 67 % of patients develop psoriasis before arthritis, and for approximately 16 % of patients, arthritis and psoriasis present within 12 months of each other [30]. The remainder—approximately 17 %—develop arthritis before psoriasis [15]. On average, the onset of inflammatory arthritis tends to occur between 7 to 10 years after the onset of psoriasis [3]. Thus, studies have demonstrated an increasing cumulative incidence of PsA with increasing duration of psoriatic disease [31•].

Age and Gender Distribution

The mean age at onset of PsA is during approximately the fourth decade [31•, 32–38]. PsA affects men and women approximately equally [3]; the percentage of affected females ranged from 38.4 % to 60 % in the most recent studies [31•, 32–39].

Epidemiology of Psoriatic Arthritis

Incidence and Prevalence of PsA

Previous studies from various parts of the world have suggested a wide variation in the incidence and prevalence of PsA in the general population. Systematic reviews in 2008 and 2012 identified studies from several different countries from their literature search [40, 41]. In these reviews, the incidence of PsA varied from 0.1 to 23 cases per 10^5 inhabitants (median 6.4 cases). Prevalence estimates varied from 1 case per 10^5 inhabitants in Japan to 420 cases per 10^5 inhabitants in Italy. Of note, these studies used different definitions for PsA, including the European Spondylarthropathy Study Group (ESSG) criteria, CASPAR criteria, diagnostic codes for PsA in the medical record, and coexisting psoriasis and arthritis, likely contributing to the observed variation. Additional studies have subsequently published results comparable to what were noted in these reviews [32, 33, 42, 43].

Estimates of the prevalence of PsA among patients with psoriasis have varied from 6-39 % [16, 34, 44–47]. The highest estimates of PsA among patients with psoriasis are generally derived from dermatology clinics, possibly due to the increased severity of psoriasis in the dermatology clinic relative to the general population. Less is known about the incidence of PsA among patients with psoriasis. A retrospective study from Germany reported that the incidence of PsA among psoriasis patients remained constant (74 per 1,000 person-years), whereas the prevalence increased with time since diagnosis of psoriasis, reaching 20.5 % after 30 years [48]. Another retrospective study from Minnesota found a lower cumulative incidence of 3.1 % of cases of PsA among psoriasis patients after 10 years from the onset of skin disease [31•]. The first study to prospectively assess the incidence of PsA among psoriasis patients was conducted in Toronto recently; 313 psoriasis patients who had at least 1 year of follow-up were included in the analysis. The annual incidence rate was found to be 1.87 (95 % confidence interval (CI) 0.71-3.03) PsA cases per 100 psoriasis patients [3].

Table 2 CASPAR criteria

A patient with inflammatory arthritis, spondylitis, or enthesitis and ≥3 points from the following elements

Feature	Points
Current psoriasis	2
History of psoriasis (unless current psoriasis was present)	1
Family history of psoriasis (unless current psoriasis or a history of psoriasis was present)	1
Dactylitis	1
Juxta-articular new bone formation	1
Rheumatoid factor negativity	1
Typical psoriatic nail dystrophy	1

Risk Factors for Developing Psoriatic Arthritis Among Patients with Psoriasis

Risk factors for the development of PsA may help to identify patients with psoriasis who should be screened for PsA or watched carefully for the development of joint symptoms. Early identification of patients at risk, in addition to lifestyle modifications, could help to prevent future joint damage and functional impairment [39]. Several noteworthy studies have addressed risk factors for PsA and have suggested that psoriasis severity, nail dystrophy [31•], smoking [49•], trauma [50], obesity [51•], obesity at the age of 18 years [52•], and prior glucocorticoid use [53] are risk factors for PsA. However, note that these findings are associations and do not necessarily reflect causation.

Smoking

A number of studies have examined smoking as a risk factor for the development of psoriasis and psoriatic arthritis, although conflicting evidence exists. Although previous

studies have suggested that smoking is a risk factor for the development of psoriasis [54], the association with the development of PsA is less clear. Two studies (notably within the same cohort) suggest that there is an inverse association between smoking and PsA among patients with psoriasis (e.g., smoking is protective, similar to findings in ulcerative colitis) [55•, 56•]. However, another study suggests that smoking is positively associated with development of PsA [49•]. In this latter study, 157 incident PsA cases were identified. Compared with never smokers, the relative risk (RR) for developing PsA was 1.54 for past smokers (95 % CI 1.06-2.24) and 3.13 for current smokers (95 % CI 2.08-4.71). With increasing smoking duration or pack-years, the risk of PsA increased monotonically (p for trend <0.0001). The population in this study was all women in the Nurses Health Study II cohort (not patients with psoriasis as in the previous studies). In this case, one may hypothesize that smoking drives psoriasis, which then drives PsA, and thus psoriasis is a mediator in the relationship between smoking and PsA. However, the results persisted even after restricting the cohort to women with a prior diagnosis of psoriasis. Smoking may play a role in the development of PsA by inducing oxidative stress that leads to an imbalance of oxidants and antioxidants resulting in chronic inflammation of the joints [57, 58]. Smoking also adversely alters the immunologic and inflammatory processes, which could contribute to the development of PsA [59, 60]. On the contrary, one theory to explain the inverse association between smoking and PsA is through the activation of the nicotinic receptor. Nicotine can activate the $\alpha 7$ nicotinic acetylcholine receptor that inhibits intracellular proinflammatory pathways that are associated with the development of arthritis [61].

Severity of Psoriasis

Several cross-sectional studies have shown that the prevalence of PsA is increased among patients with severe psoriasis [31•, 62]. This may make sense biologically but also may represent observation bias in that patients with more extensive psoriasis may be followed more closely by physicians and therefore are more likely to have their inflammatory arthritis come to medical attention. Although studies suggest an increasing prevalence of PsA with increasing psoriasis severity, at any one time point, the disease activity in the skin and joints does not necessarily correlate (e.g., one can be flaring while the other is stable). Additionally, it is important to recognize that the majority of patients with PsA have mild psoriasis and a relatively small proportion of patients with psoriasis have severe disease [63].

Nail Dystrophy

Typical psoriatic nail dystrophy is a known risk factor for development of PsA [31•]. Recent studies have shown that nail changes are more frequent in patients with PsA compared with those with psoriasis alone [14, 56] and that nail changes were more frequent in the PsA patients with distal interphalangeal joint (DIP) involvement than those without DIP involvement (63 % vs. 41.2 %, $P<0.05$) [15]. Interestingly, an ultrasound study in 2012 showed that nail involvement in psoriasis also is associated with a higher systemic enthesopathy score [64•]. It is unclear why nail dystrophy may be associated with a higher risk of PsA. It is possible that dystrophic nails represent a marker for immunoreactivity, leading to PsA in a subset of psoriasis patients. A study by Scarpa and colleagues noted that nail involvement is present in almost all PsA even if it is not clinically evident. Furthermore, given the close proximity of the DIP joint to the nail matrix, they postulated that DIP joint involvement may actually occur as a result of nail disease [65]. This study raises the possibility that nail dystrophy is an indicator of ongoing DIP involvement.

Location of Psoriasis

Wilson et al. found that patients with psoriasis involving the intergluteal and/or perianal areas and psoriasis involving more than three affected areas had a higher risk of developing PsA, with a hazard ratio (HR) of 2.35 (95 % CI, 1.32-4.19) and HR 2.24 (95 % CI, 1.23-4.08), respectively [31•]. In addition, studies have shown that scalp psoriasis is seen more frequently in PsA patients compared with patients with psoriasis only [15, 31•]. Some argue, however, that because the scalp is commonly one of the first areas affected by psoriasis [66] that patients with more severe psoriasis (and likely a higher chance of developing PsA as discussed above) will inevitably have more scalp disease.

Obesity

Obesity has been associated with an increased risk of psoriasis [67], and more recent studies have found associations between obesity and PsA. In cross-sectional studies, PsA patients had a higher body mass index (BMI) than healthy controls [68, 69] and PsA was more prevalent among obese psoriasis patients than nonobese psoriasis patients [62]. The directionality of this association is still relatively unclear. Is it that patients with PsA have difficulty with mobility and thus become obese or does obesity predispose the patient to development of inflammatory arthritis because of biochemical or mechanical factors? Studies to date suggest the former [70, 71], but the relationship is confusing given that the incidence of joint pain and osteoarthritis is more prevalent among obese individuals [72], creating a potential for misclassification bias. However, a recent cohort study found that the incidence of PsA increased with increasing BMI. Compared with psoriasis patients with BMI <25 kg/m^2, the

RRs for developing PsA were 1.09 (0.93-1.28) for BMIs from 25.0 to 25.9, 1.22 (1.02-1.47) for BMIs from 30.0 to 34.9, and 1.48 (1.20-1.81) for BMIs ≥35 [50].

Markers of Progression in Patients with Psoriatic Arthritis

Although psoriatic arthritis was once thought to be a milder disease than rheumatoid arthritis, we now know that it can be quite destructive with nearly 20 % of patients disabled by their disease [73]. In addition, this destruction can happen quite early in the disease course; approximately half develop radiographic damage in a median of 2 years after symptom onset [74]. Although disease course is as variable as the clinical phenotypes of PsA, the number of actively inflamed joints (and particularly swollen joints) is associated with both radiographic damage and clinically apparent damage, defined as decreased range of motion of the joint, joint deformities, loosening, or ankylosis [75, 76]. Furthermore, tenderness and swelling of a joint can predict damage to that particular joint [77]. Simon et al. also found that an increasing joint count over the course of the retrospective study correlated with radiographic progression [76]. Finally, among the placebo group in a phase III clinical trial (ADEPT), elevated CRP at baseline was a risk factor for radiographic progression [78].

Identification of Early Psoriatic Arthritis

Previous reports have suggested that approximately 10 % of patients with psoriasis followed in a dermatology clinic have undiagnosed PsA, although this may be an underestimate. Haroon et al. recently reported that, after excluding patients with known PsA, 29 % patients with psoriasis systematically assessed for PsA had the disease [17]. Ibrahim et al. found that among 93 patients with psoriasis, 12 had PsA and of these, only 4 had a previously known diagnosis of PsA [79]. Not only is PsA underdiagnosed, but the diagnosis often is delayed when made. Given the high burden of radiographic disease even early in the disease course [74], it is important to identify patients with PsA early in their course because early initiation of treatment may improve the disease course. Although evidence for early treatment initiation does not yet exist in PsA [80], in RA early initiation of therapy (e.g., methotrexate) results in improved response to therapy and decreased erosions at 2 years compared with those with later treatment initiation (>3 months after symptom onset) [81–84]. A recent observational study by Gladman et al. provided support for this concept; patients who presented after 2 years of symptoms had a much greater rate of progression than those who presented earlier in the disease course [85]. Dermatologists play a key role in identification of PsA, because patients often are unaware of the association between their skin condition and joint symptoms.

Screening Tools

Whereas dermatologists will have varying comfort in assessing for PsA, simple screening tools have been developed to help identify which patients should receive further evaluation for the diagnosis of PsA. Four screening tools have been developed to date (Table 2): the Psoriasis Epidemiology Screening Tool (PEST), Toronto Psoriatic Arthritis Screening (ToPAS), Psoriasis and Arthritis Screening Questionnaire (PASQ), and Psoriatic Arthritis Screening and Evaluation (PASE). These tools were designed specifically to help dermatologists identify those patients with psoriasis who would benefit from a prompt referral to rheumatology. All are one- or two-page questionnaires that can be easily administered during office visits and performed relatively similarly, with sensitivities ranging from 76 % to 92 % and specificities ranging from 73 % to 93 % in the original validation studies [86–90]. An electronic version of the PASQ tool (ePASQ) was recently created and is available for download on the Ipad and Iphone at the App store [91]. There is no preference for one tool over another. Of note, a positive screening questionnaire does not mean the patient has PsA. Mimics of PsA, such as osteoarthritis, fibromyalgia, gout, pseudogout, and RA, also may screen "positive."

It is important to recognize that these screening questionnaires, while they can be helpful, have been found to have much lower sensitivity and specificity than those reported in the initial validation studies, particularly when applied among psoriasis patients without known PsA (the original validation studies included patients with known PsA) [88–90]. Compared with a rheumatologist's assessment ("gold standard"), the sensitivities of these instruments ranged from 24-41 % and specificities ranged from 90-98 % in a recent study by Haroon et al. among patients with psoriasis but without known PsA. Positive and negative predictive values also were lower than previous described: 63-88 % and 75-82 % respectively [17]. Walsh et al. performed a similar study and found sensitivity 60-76 % and specificity 41-55 % after excluding those with known PsA [92]. The results of these recent studies question the usefulness of the existing questionnaires and suggest the need for the development of new instruments.

Evaluation of the Patient with Psoriasis and Joint Pain

The differential diagnosis for joint pain in a patient with psoriasis is broad. As mentioned earlier, it includes psoriatic arthritis, osteoarthritis, fibromyalgia, gout, pseudogout, RA, other spondyloarthropathies, overuse syndromes, and mechanical- and trauma-related joint pain. Historical elements suggestive of inflammatory arthritis include severe and persistent pain for >6 weeks, morning stiffness (or

stiffness after prolonged rest) lasting >30 minutes, systemic symptoms, such as fatigue, and swelling of the joints [93, 94]. History of dactylitis or swelling of the entire digit such that it appears like a "sausage" is highly suggestive of psoriatic arthritis. Quick screening questions for inflammatory arthritis include the presence of joint pain, prolonged morning stiffness, and joint swelling.

Physical examination is still the "gold standard" for diagnosing inflammatory arthritis. Examination features are noted in Table 1. Synovitis has been described as a "spongy" feeling as if there is a "layer of bread dough between the skin and bony margins of the joint [95]." This is distinct from bony enlargement and crepitus (crackling feeling while moving the joint through the range of motion) felt on examination of the osteoarthritic joint. Tenderness over the joint lines can be suggestive of inflammation, although this can be present in the other conditions mentioned as well. Examination of the entheses is best accomplished by applying pressure over the area where the tendon inserts onto the bone and assessing for tenderness. For example, pressure is applied over the region where the Achilles tendon inserts onto the calcaneous. Inflammation in the entheses can be difficult to visualize. The Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA) has developed videos to teach dermatologists examination techniques for identifying PsA. These are currently only available to GRAPPA members but will soon be widely available (the web address was not available at the time of publication) [96].

No laboratory biomarkers are available for diagnosing PsA, and there are not specific recommendations for laboratory workup of suspected inflammatory arthritis, because this is usually driven by the clinical history [97]. An elevated C-reactive protein (CRP) and/or sedimentation rate may suggest systemic inflammation. However, these inflammatory markers can be normal in approximately half of patients with active PsA and can be elevated in patients with psoriasis but without arthritis. Rheumatoid factor (RF) and anti-cyclic citrullinated peptide (CCP) can be used to evaluate for RA in the setting of polyarthritis, although 10-20 % of patients with PsA will have a positive RF. Anti-CCP also can be positive in PsA. A negative RF is helpful, because it satisfies an element of the CASPAR criteria.

There is no consensus on the need for imaging studies in diagnosis and/or monitoring of PsA. Plain film radiographs (x-rays) of the joints involved can be useful if new bone formation, erosions, osteolysis, or ankylosis are present [98]. However, early in the course of the disease, these findings most often will not be apparent. Musculoskeletal ultrasound (US) is increasingly used to aid in diagnosis. When there is a question about the examination a joint or enthesis, US can be helpful to establish whether or not inflammation is present. US detects entheseal abnormalities, including tendon edema and thickening, tendon tears, bone erosions, enthesophytes, bursitis, and increased blood perfusion via power Doppler [99]. Magnetic resonance imaging (MRI) is useful to assess the spine and sacroiliac joints, because inflammation in these areas often is missed on x-rays [100].

Although there is no consensus for which patients should be referred to rheumatologist and what initial workup should be performed, we propose the algorithm shown in Figure 1. One of the difficulties when diagnosing inflammatory arthritis is that not all joint pain represents inflammation and the reasons for joint pain can be very difficult to distinguish, even for experienced rheumatologists. Therefore, we strongly recommend that all patients suspected of having inflammatory arthritis be seen by a rheumatologist for confirmation of the diagnosis and therapy initiation. After the diagnosis has been confirmed, the rheumatologist and dermatologist should work together to manage the patient's symptoms.

Treatment of Psoriatic Arthritis

A full discussion of treatment is beyond the scope of this review. The treatment of psoriatic arthritis can be complex, because it often is tailored to the manifestations of the disease. For example, spondylitis, dactylitis, and enthesitis often respond best to TNF-alpha inhibitors rather than traditional disease modifying antirheumatic drugs. We suggest referring to the GRAPPA guidelines or the European Union League Against Rheumatism (EULAR) treatment recommendations [101–104]. However, it is important to note that these guidelines are in flux given recent evidence suggesting that methotrexate may not act as a disease modifying agent in the treatment of PsA [105, 106]. Furthermore, there are several new drugs in the pipeline for the treatment of PsA.

Other Considerations

Cardiovascular Disease

Multiple studies have now demonstrated the increased risk of cardiovascular disease (CVD) in patients with psoriasis but fewer studies specifically examine PsA. These studies suggest that patients with PsA also are at an increased risk for developing CVD [107–109]. CVD is among the leading causes of death in patients with PsA [110, 111]. Furthermore, traditional cardiovascular risk factors (hypertension, diabetes, and hyperlipidemia) as well as peripheral vascular disease, congestive heart failure, atherosclerosis, and ischemic heart disease are more prevalent among patients with PsA than controls [107]. Patients with PsA may be at increased risk for developing diabetes [112]. A recent study examining the association between PsA and CVD in a large cohort of U.S. women found

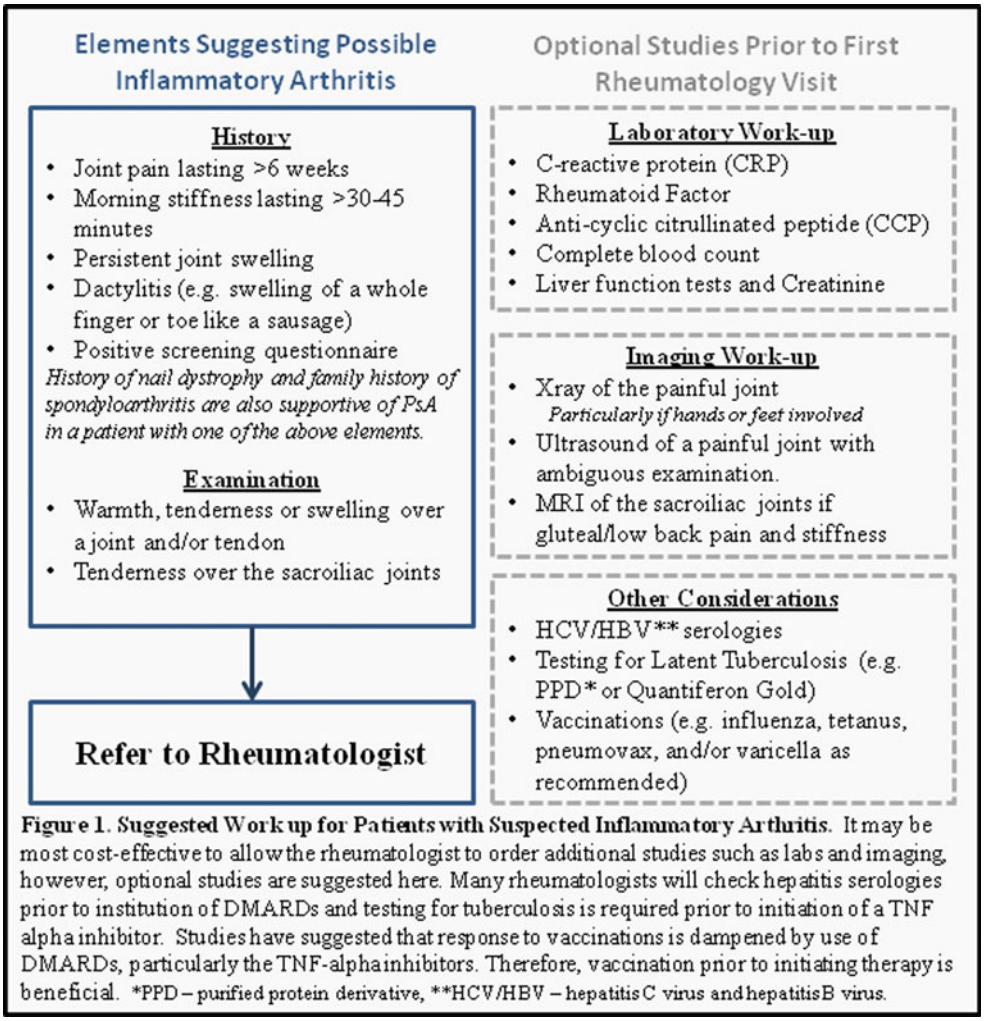

Figure 1. Suggested Work up for Patients with Suspected Inflammatory Arthritis. It may be most cost-effective to allow the rheumatologist to order additional studies such as labs and imaging, however, optional studies are suggested here. Many rheumatologists will check hepatitis serologies prior to institution of DMARDs and testing for tuberculosis is required prior to initiation of a TNF alpha inhibitor. Studies have suggested that response to vaccinations is dampened by use of DMARDs, particularly the TNF-alpha inhibitors. Therefore, vaccination prior to initiating therapy is beneficial. *PPD – purified protein derivative, **HCV/HBV – hepatitis C virus and hepatitis B virus.

Fig. 1 Suggested workup for patients with suspected inflammatory arthritis. It may be most cost-effective to allow the rheumatologist to order additional studies, such as labs and imaging; however, optional studies are suggested here. Many rheumatologists will check hepatitis serologies before institution of DMARDs and testing for tuberculosis is required before initiation of a TNF-alpha inhibitor. Studies have suggested that response to vaccinations is dampened by use of DMARDs, particularly the TNF-alpha inhibitors. Therefore, vaccination before initiating therapy is beneficial. PPD, purified protein derivative; HCV/HBV, hepatitis C virus and hepatitis B virus

a significantly elevated risk of nonfatal CVD, particularly MI, in women with PsA, even after adjusting for traditional cardiovascular risk factors. Adjusted hazard ratios (95 % CI) for nonfatal CVD, nonfatal myocardial infarction (MI), and nonfatal stroke were 5.32 (2.85-9.94), 7.4 (3.5-15.66), and 3.18 (1.02- 9.91) respectively [113••]. Even patients with early PsA (diagnosis within 2 years) seemingly incur this risk [114]. Health care providers should be aware of the increased risk for CVD in patients with PsA, counsel patients on these comorbid conditions, and refer to primary care or cardiology specialty care accordingly for management.

Summary

- PsA can be monoarticular, oligoarticular, or polyarticular inflammatory arthritis. Additional features include spondylitis, arthritis mutilans, enthesitis, and dactylitis.
- The CASPAR criteria are classification criteria most often used to define PsA in research studies, and although designed for the purpose of research, are most helpful in clinical practice.
- There is a wide variation of incidence and prevalence of PsA in the world, and prevalence of PsA in psoriasis ranges from 6-39 %. This is likely due, in part, to varied study designs and definitions of PsA.
- The average age of onset for PsA is in the mid 40s. PsA most often occurs years after the onset of psoriasis but can occur before or at the same time as the onset of clinically apparent skin disease.
- Potential risk factors for PsA include nail involvement, obesity, severity/location of psoriasis, and smoking.
- There are four main screening tools for PsA: (PEST), (ToPAS), (PASQ), and (PASE). These can be used easily in an office setting to determine which patients would benefit from a referral to a rheumatologist.
- Physicians who treat individuals with psoriasis and PsA should address modifiable CVD risk factors. These include smoking, obesity, hypertension, hyperlipidemia, and diabetes.

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- • Of importance
- •• Of major importance

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