Psoriatic Arthritis

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Overview of Psoriatic Arthritis

- Psoriatic arthritis (PsA) is a distinct form of inflammatory arthritis that is largely, but not always, associated with cutaneous psoriasis, making psoriasis one of the most important biomarkers in rheumatology.
- The prevalence of PsA in the general population is estimated to be between 0.5% and 1%, only slightly less common than that of rheumatoid arthritis (RA). Up to 30% of people with psoriasis will develop PsA.
- PsA is a member of the spondyloarthritis family of diseases, along with axial spondyloarthritis (including ankylosing spondylitis), reactive arthritis, and the arthropathy of inflammatory bowel disease.
- A variety of clinical patterns of PsA are recognized: distal joint disease, oligoarthritis (less than five joints), a symmetrical polyarthritis, and spondylitis. Some patients present with combinations of these features, e.g., oligoarthritis plus spondylitis. In addition, people may have co-existing enthesitis and dactylitis (sausage digit), clinical features that are of considerable help in making the diagnosis.
- These clinical patterns are not necessarily stable over time. For example, distal joint disease can evolve into polyarthritis, and axial disease develops in up to 40% of patients who present with other patterns of arthritis involvement.
- The most common pattern of joint involvement at presentation is polyarthritis, followed closely by oligoarthritis. Approximately 45% of cases have an oligoarthritic pattern at presentation.

Myth *The presence of anti-citrullinated protein antibodies (ACPA) in association with peripheral synovitis indicates rheumatoid arthritis, even in the presence of psoriasis.*

Reality: Most patients with early-onset arthritis are screened for autoantibodies, including ACPA, as these are important in the differential diagnosis of arthritis. Many doctors assume that the presence of these antibodies indicates that the patient has rheumatoid arthritis. However, up to 7% of people with PsA have ACPA at low titers ACPA (Taylor et al. 2006), and just over 10% may have rheumatoid factor (Gladman et al. 1987). The diagnosis ultimately rests on careful clinical examination and investigations.

Patients with PsA who have ACPA also have unequivocal clinical signs of PsA–dactylitis, enthesitis, and axial disease in some cases, as well as typical radiology findings such as entheseal new bone formation and osteolysis (Helliwell et al. 2007). The principal differences between PsA and RA are summarized in Table 8.1. It is important to recognize the presence of skin (and nail psoriasis (Fig. 8.1)). Psoriasis is one of the best biomarkers we have in rheumatology. If someone walks into your consulting room with a patch of psoriasis on the elbow and a swollen finger, the likelihood of a PsA diagnosis is high.

Extra-articular features of PsA and RA also contrast strikingly: urethritis and inflammatory bowel disease symptoms are common in PsA, but interstitial lung disease, myocarditis, and pericarditis are far more characteristic of RA. The extra-articular differences are also found in the eye. Anterior uveitis (iridocyclitis) is typical of PsA, but episcleritis, scleritis, keratitis, and keratoconjunctivitis are much more likely to complicate RA.

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Table 8.1 Contrasts between psoriatic and rheumatoid arthritis

Disease feature	Psoriatic arthritis	Rheumatoid arthritis
Nail lesions	Pitting	Bywaters' lesions
	Onycholysis	Splinter hemorrhages
Joint pattern in hands and wrists	Fewer joints involved leading to asymmetry	More joints leading to symmetry
	DIP involvement common	MCPs/PIPs
	"Ray" pattern	Ulnar styloid involvement
	Dactylitis ("sausage" digits)	
Spinal disease	Syndesmophytes	C1-C2 atlanto-axial inflammation and subluxation
Eye disease	Anterior uveitis	Episcleritis
		Keratoconjunctivitis
		Scleritis
		Peripheral ulcerative keratitis
Other extra-articular features	Cutaneous psoriasis	Nodules
	Bluish discoloration over joints	Interstitial lung disease
	Enthesitis, especially of the Achilles insertion, knee, and lateral epicondyle of the elbow	Pericarditis
	Urethritis	Systemic vasculitis
	Inflammatory bowel disease	
Antibodies	Low titer ACPA in 7%	ACPA in 60–70%

Pearl *PsA tends to occur in a "ray" distribution.*

Comment: PsA has a tendency to affect all joints in a particular digit, e.g., the MCP, PIP, and DIP joints of the same finger. If the soft tissues are also involved, the clinical picture is one of dactylitis, or "sausage digit" (in the toes, these are called chipolatas!) (Fig. 8.2a, b). The sausage appearance of the affected digit results from the involvement not only of the joints by synovitis but also from tenosynovitis, particularly of the flexor tendons, and soft-tissue edema. In fact, dactylitis is a pathophysiological paradigm of PsA—inflammation is seen in joints, tendons, entheses, bone, and soft-tissue, all in the same body region (Healy et al. 2008).

Dactylitis is more common in the toes than in the fingers and in *particular* the second and fourth toes. Dactylitis can be hard to identify in the first and fifth toes, as both of those digits can look a little swollen at the best of times. Using the opposite side for comparison helps. In some patients, dactylitis becomes chronic such that the finger remains swollen but is no longer painful or red, though in these cases some inflammation in the tissues persists.

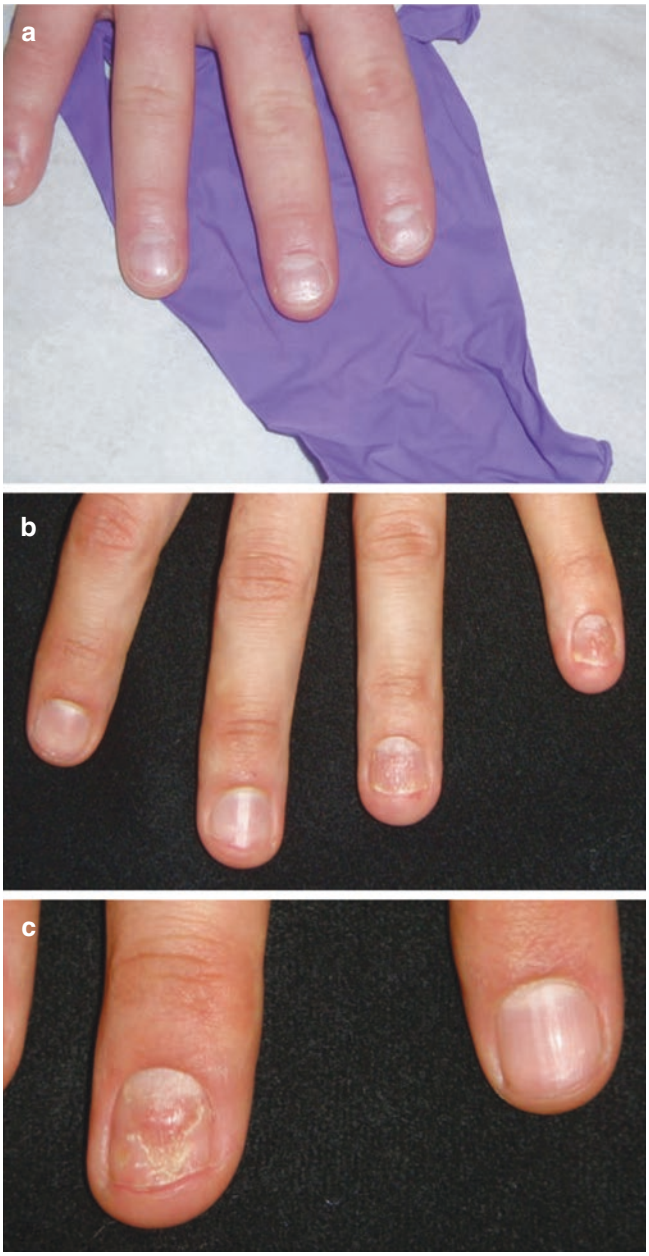

Fig. 8.1 Nail changes in psoriasis. (**a**) Nail pitting. (**b**) Onycholysis. (**c**) Close view of onycholysis

Pearl *Rheumatoid nodules do not occur in PsA.*

Comment: True rheumatoid nodules do not occur in PsA. But this distinction is less helpful than it once was, for the following reasons. First, rheumatoid nodules are becoming much less frequent in clinical practice. The likely explanations for that are that rheumatoid nodules occur in seropositive RA patients who smoke, and smoking is less common than in earlier eras. Second, early and aggressive treatment may prevent rheumatoid nodules from developing in RA.

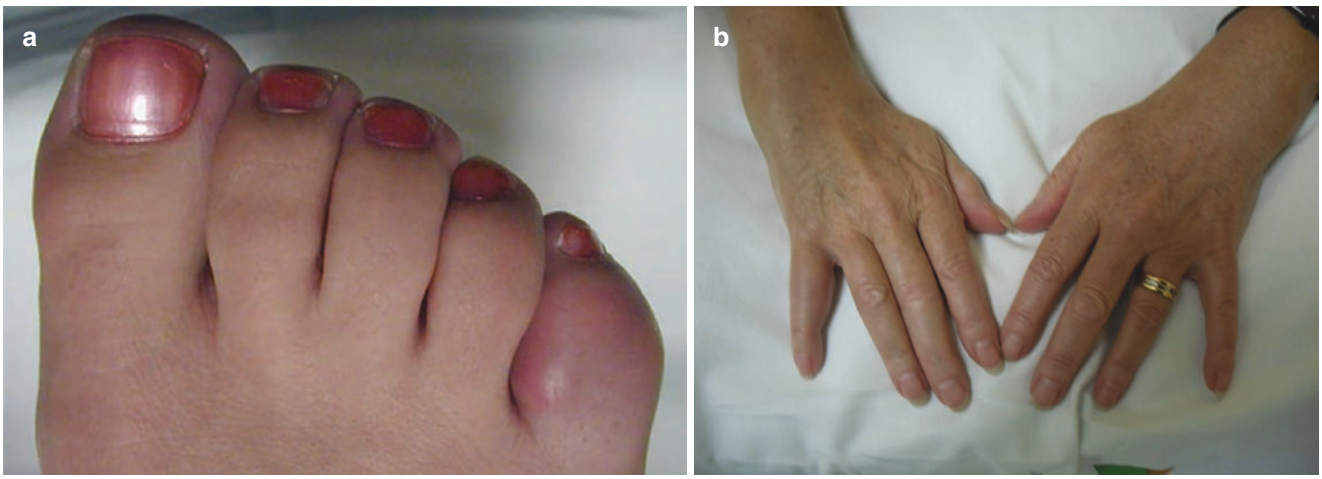

Fig. 8.2 Dactylitis of the hand and foot. (**a**) Dactylitis of the little toe. (**b**) Dactylitis of the right fifth finger (and synovitis of the right wrist)

However, PsA patients may develop nodules for other reasons. Concomitant gout and PsA are common, and patients with chronic gout may present with both nodular olecranon bursae and finger tophi that might be mistaken for rheumatoid nodules. If in doubt, aspirate, and look for uric acid crystals. Radiography may help—look for classic "rat bite" erosions of gout.

Pearl *Inflammatory arthritis in PsA can occur before cutaneous psoriasis appears.*

Comment: Approximately 30% of patients with psoriasis will develop PsA. The majority of patients with PsA have psoriasis either simultaneously or before the onset of arthritis. However, approximately 15% of PsA patients have arthritis that precedes their cutaneous disease. In those patients, the clinical pattern of joint involvement and the other musculoskeletal features are critical to arriving at the correct diagnosis (Table 8.1). It is at such times that a careful history, family history, and physical examination are crucial in making the diagnosis.

Sometimes the skin disease is hidden. Always check the scalp, particularly the hairline (Fig. 8.3a, b) and behind the ears. Check the armpits, the umbilicus, and the natal cleft for flexural psoriasis. Examine the nails for subtle changes of onycholysis and pitting (Fig. 8.1). Patients often present with the musculoskeletal features of PsA but only a tiny patch of psoriasis—often insignificant to the patient—or just a history of psoriasis that is apparently no longer active.

Myth *There is often no direct relationship between the skin and the musculoskeletal disease in PsA.*

Reality: Some investigators suggest that there is a relationship between the severity of psoriasis and the prevalence of PsA; i.e., the more severe the skin disease, the higher the likelihood of inflammatory joint disease (Gelfand et al. 2005). However, once a patient has developed PsA, there does *not* appear to be a direct relationship between the flares of skin and joint disease (Jones et al. 1994; Elkayam et al. 2000).

Some medications employed by rheumatologists exacerbate cutaneous psoriasis. These include nonsteroidal anti-inflammatory drugs and glucocorticoids. Moreover, some medications can alter the phenotype of the psoriasis. TNF inhibitors, for example, may induce palmoplantar pustulosis. When cutaneous psoriasis is a significant part of psoriatic disease, consultation and co-management with a dermatologist is recommended (Warren et al. 2013).

Pearl *Spinal involvement presents later in life in PsA than it does in radiographic axial spondyloarthritis (i.e., ankylosing spondylitis (AS)) and is often less symptomatic.*

Comment: Patients with AS present with back pain in their late teens or early 20s. In contrast, those with psoriatic spondylitis seldom present before their 30s or 40s. Moreover, the presenting feature of spondylitis in PsA is often the limitation of movement rather than back pain. Just as the peripheral arthritis of PsA is less symptomatic than that of RA, so too is the spinal disease of PsA frequently less symptomatic

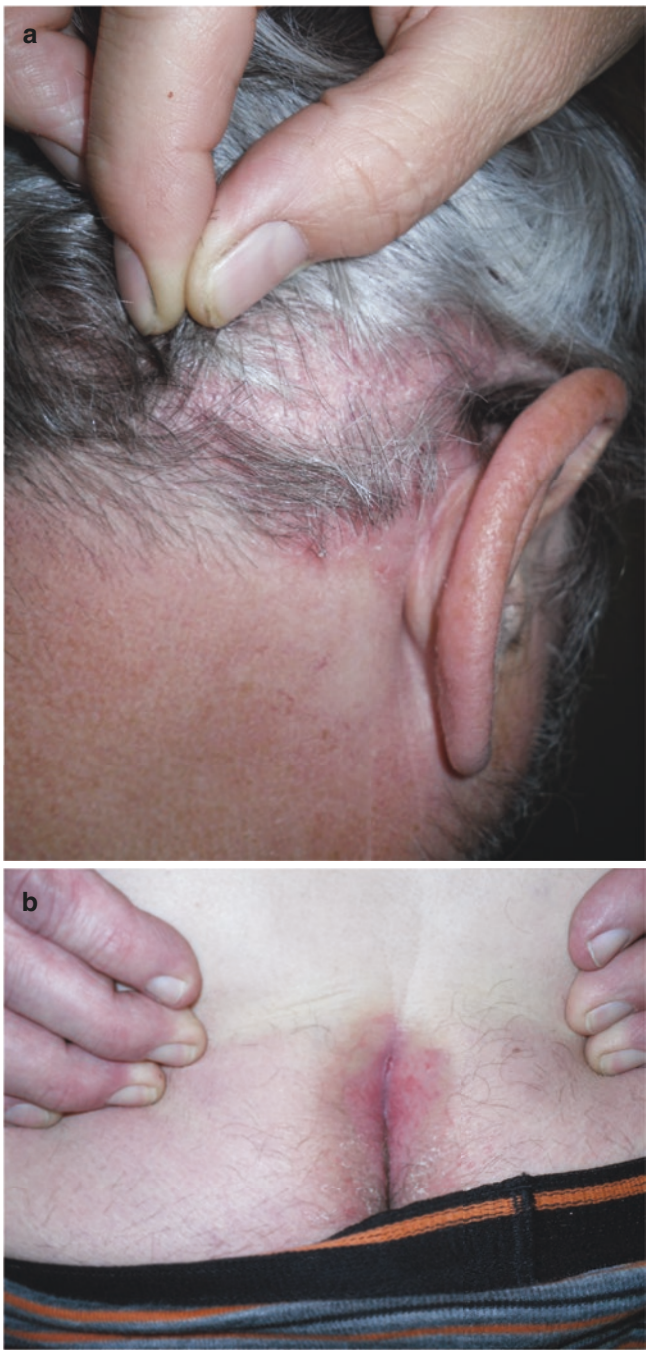

Fig. 8.3 "Hidden" psoriasis. (**a**) Behind the ear. (**b**) At the natal cleft

that of AS (Gladman et al. 1993; Helliwell et al. 1998). Patients with PsA often demonstrate more spinal mobility on average than do AS patients, a consequence of less structural damage.

It is worth noting, however, that axial involvement in PsA can present as either classical AS or as an alternative phenotype. The alternative phenotype demonstrates less severe and less symmetric structural change. The two phenotypes are likely driven by genetics, with the classical phenotype being driven by the presence of HLA-B27 (Helliwell 2020) (Fig. 8.4).

Myth *PsA does not require aggressive intervention.*

Reality: This notion was prevalent following the early descriptions of PsA. Verna Wright carried out the key early clinical characterization of PsA and, together with John Moll, produced the first diagnostic/classification criteria (Moll and Wright 1973). Wright observed that PsA tends to be less severe than RA (Wright 1959), and a later review of the cohort in Leeds seemed to confirm this (Roberts et al. 1976). Other cohorts from North America also supported this notion (Shbeeb et al. 2000; Coulton et al. 1989).

More recent studies, however, have confirmed the high prevalence of severe, destructive, and disabling disease in PsA, as well as identifying an increased mortality risk for patients with this diagnosis (Gladman et al. 1998; Sokoll and Helliwell 2001; Bond et al. 2007). A large proportion of PsA patients develop joint erosions and disability within the first 2 years of disease. Early, aggressive treatment of PsA may prevent untoward outcomes. There is no doubt that outcomes are improving in PsA, probably because of improved treatments (Ali et al. 2007).

Myth *PsA is basically a disease of the skin and joints.*

Reality: PsA is associated with significant comorbidities. There are significant associations with obesity, the metabolic syndrome, and diabetes mellitus. Patients are at an increased risk for coronary artery disease and atherosclerosis elsewhere, and demonstrate increased mortality as a result of this. There are significant associations with inflammatory bowel disease, inflammation in the anterior eye (iritis), and depression (Eder et al. 2013; Tam et al. 2008; Boehncke et al. 2011).

Pearl *Gout and PsA can occur in the same patient.*

Comment: Gout and PsA do frequently co-exist and it may be hard to distinguish the contribution of each disorder to the overall burden of arthritis. Hyperuricemia is a common finding in psoriasis and clinical gout may result partly as a consequence of psoriasis, perhaps because of the increased cell turnover that marks cutaneous psoriasis.

It is particularly important to consider gout when the patient presents with intermittent oligoarthritis. By all means, check the serum uric acid. But also examine the patient for tophi (don't want to overlook those!) try to aspirate an acutely inflamed joint and examine the synovial fluid for

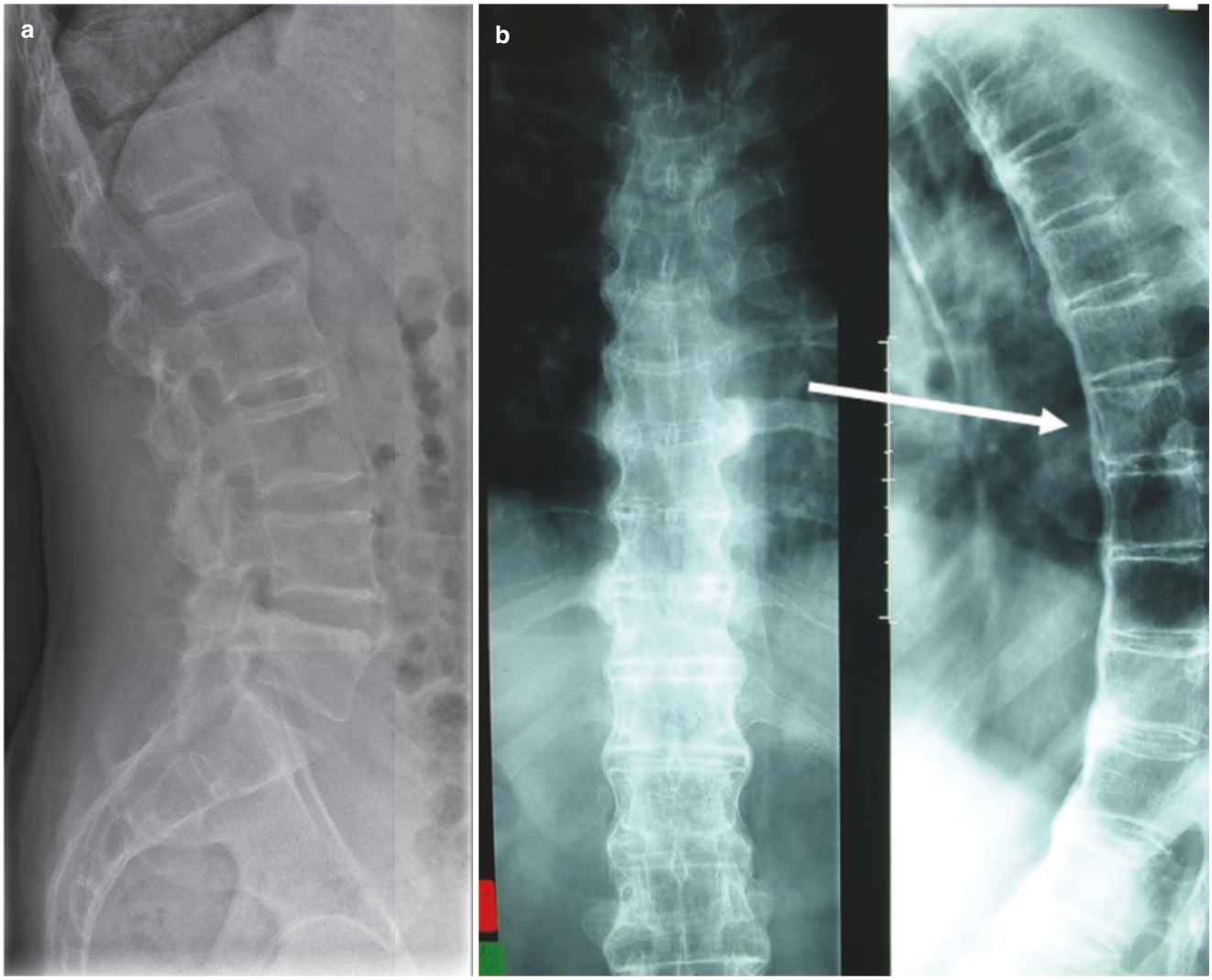

Fig. 8.4 Axial changes typical of psoriatic axial spondyloarthritis. (**a**) Non-marginal syndesmophytes. (**b**) Para-vertebral ossification. (**c**) Para-vertebral ossification shown from lateral view

urate crystals. A dual-energy computed tomographic (DECT) scan seeking to identify urate deposition in the tissues can also be useful. In early cases, ultrasound may help by demonstrating the double contour sign. In longstanding cases, plain radiographs may help, showing typical "rat bite" erosions associated with gout.

Myth *Methotrexate is not effective for enthesitis and dactylitis in PsA.*

Reality: Methotrexate generally retains its position as the first disease-modifying drug given to patients with PsA. The advantages of this drug are that it is cheap, familiar to rheumatologists, and works for both the skin and joints. In fact, improvement in cutaneous psoriasis is usually seen early, and that makes both the patient and rheumatologist convinced that the drug is working. However, due to the fact that the drug is now non-branded, there has been no incentive to do properly conducted randomized clinical trials in PsA, examining efficacy across the clinical spectrum of disease. Therefore, learned societies developing treatment guidelines have not felt confident in recommending it for clinical features such as enthesitis and dactylitis (Coates et al. 2016; Singh et al. 2019). However, clinicians do see improvement with methotrexate overall, and two open-label studies conducted recently suggest methotrexate may be effective for these disease manifestations (Coates and Helliwell 2016; Mease et al. 2019). There are still no efficacy data about the use of methotrexate for axial disease.

Pearl *PsA can present as an indolent disease with manifestations occurring over several years.*

Comment: Rheumatologists are asked to evaluate the more severe end of the PsA spectrum, when patients need referral and treatment. However, community-based studies reveal a different picture: that of a fluctuating, slowly-evolving disease. People may report intermittent musculoskeletal symptoms over several years, such as plantar fasciitis that eventually settled; a swollen knee that appeared to develop after a fall or twist; or a dactylitic finger or toe that lasted days or weeks and resolved spontaneously or responded to over-the-counter NSAIDs. These patients may never present to rheumatologists at tertiary care centers and may not be identified by their dermatologist, either. The central role played ostensibly by trauma in some of these self-limited episodes has led to the suggestion in the past that flares of PsA may be triggered by "deep" Koebner phenomena (the Koebner phenomenon occurs when the underlying skin disease is triggered at a site, by trauma). This hypothesis remains unproven.

Myth *All patients with PsA need systemic treatment.*

Reality: Although PsA can be severe in impact and warrant early, aggressive treatment, some patients present with mild PsA that waxes and wanes over time. These patients may manage well for extended periods off all systemic therapy with no sign of joint damage. Some may only need to take systemic treatment intermittently, being able to taper and stop treatment for periods. Caution should be maintained because our ability to predict prognosis is poor. Some of these patients may develop more severe disease over time.

Myth *Treatment of PsA should primarily focus on the joint disease.*

Reality: The clinical courses of PsA patients vary, but when considering the six key domains of PsA (peripheral arthritis, axial disease, enthesitis, dactylitis, skin, and nail disease), the majority present with multiple domain involvement and not just skin only. All of these domains have a significant impact on patients' quality of life. Treatment must be tailored for individual patients such that all domains are addressed. Enthesitis and skin disease have a particularly high patient burden (Wervers et al. 2018).

Myth *Distal interphalangeal joint involvement due to PsA cannot be distinguished from osteoarthritis.*

Reality: It is sometimes difficult to distinguish between distal interphalangeal joint swelling due to osteoarthritis and that caused by PsA. Both clinical features and plain radiographs, however, can help. The typical patient with osteoarthritis is in their 50s and 60s, and osteoarthritis may be present in other joints such as the proximal interphalangeal joint, first metatarso-phalangeal joint, and knee. Often a family history of hand osteoarthritis is present. In osteoarthritis, the symptoms are often minimal and the joint feels very bony. These are Heberden's nodes in the distal interphalangeal joints and Bouchard's nodes in the proximal interphalangeal joints. In contrast, in PsA the joint may have a "squishy" component due to synovitis, and adjacent nail changes are often present (Fig. 8.5). Moreover, other small joints of the hand may be involved in PsA, and the involvement may be patchy and asymmetrical (Helliwell et al. 2000). Occasionally, all three joints in a finger may be involved: the "ray" distribution, with or without dactylitis.

Plain radiographs should help. The new bone formation adjacent to the joint margin in PsA is often "fluffy" and juxta-articular (enthesiophytes). In contrast, in osteoarthritis it is more lateral and well defined and extends outward from the joint margin (osteophytes). These patterns are often called "mouse ears" for PsA and "gull's wings" for osteoarthritis (Fig. 8.6a, b) (Taylor et al. 2003).

Pearl *Patients with psoriasis may have widespread pain, and it is sometimes difficult to distinguish polyenthesitis from fibromyalgia.*

Comment: Anatomical surface landmarks for entheseal points may be near to those points identified as indicating fibromyalgia. In particular, the lateral epicondyle of the elbow, the entheseal point for the common extensor tendon, is near to the fibromyalgia point just distally. At the knee, the entheseal point at the proximal insertion of the medial collateral ligament is near to the fibromyalgia point on the medial aspect of the knee at the joint line. Further, people with fibro-

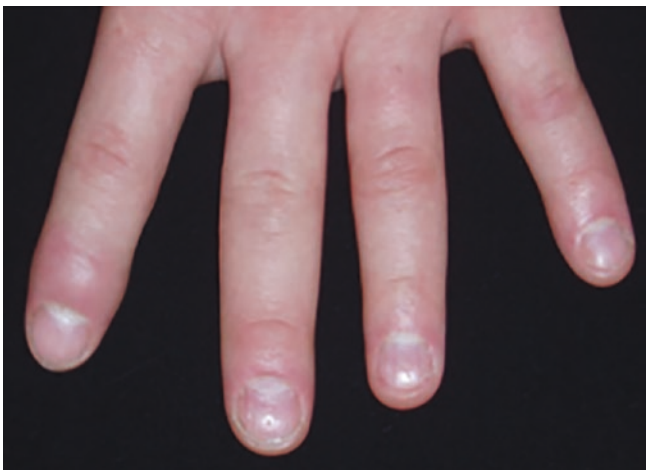

Fig. 8.5 Distal interphalangeal (DIP) joint arthritis in psoriatic arthritis (PsA). The arthritis is evident in the DIP joint of the digit shown at the far left of the figure. This patient also had nail pitting. DIP involvement and nail pitting often occur together in PsA

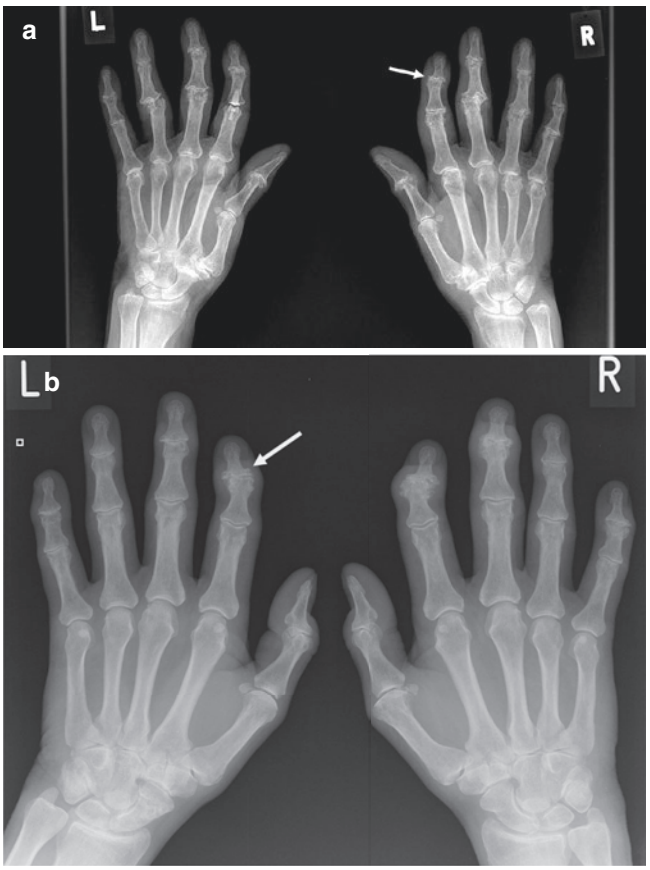

Fig. 8.6 Changes in the distal interphalangeal joints in osteoarthritis and PsA. (**a**) Osteoarthritis, demonstrates the typical "gull wing" appearance of new bone in the distal interphalangeal joint (arrow). (Image courtesy of Dr. N S Chew). (**b**) Psoriatic arthritis demonstrates typical "mouse ears" appearance of new bone formation in the second left distal interphalangeal joint (arrow) and marked periarticular new bone formation at the right second distal interphalangeal joint. (Image courtesy of Dr. M Chandramohan)

myalgia have allodynia and have increased sensitivity to pain generally, not just at the fibromyalgia points. So how do we, as clinicians, distinguish the tenderness resulting from fibromyalgia from that due to the polyenthesitis of PsA?

There are two important points here. First, patients may have *both* polyenthesitis and fibromyalgia. In fact, central sensitization and chronic, widespread pain may be one *result* of their (painful) PsA. In these cases, in patients with known PsA, look for other signs of psychological distress, a high tender to swollen joint count ratio, and fibromyalgia tender points. An important study performed in Milan, evaluating patients with fibromyalgia and PsA, identified the best ways to distinguish these two conditions (Marchesoni et al. 2012). Clinically, this is an important question: patients are often referred to rheumatologists because of multiple biologic failure, but the patients have been labelled "treatment failures" because of their associated fibromyalgia, which will never respond to disease modifying therapy. Second, ultrasound may be useful in differentiating these two conditions, but the relationship between ultrasound enthesitis and clinical enthesitis is poor. Many healthy individuals and patients with fibromyalgia have abnormal ultrasound scans of their entheses, so, here again, clinical correlation is crucial (Macchioni et al. 2019).

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