# LEAP ROUNDS

**Johns Hopkins University**
**School of Medicine**
Division of Rheumatology
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## Psoriatic Arthritis

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The Art and Science of
Personalized Treatment

# PSORIATIC ARTHRITIS

## The Art and Science of Personalized Treatment


A FINGERPRINT OF DISEASE, A MOMENT IN TIME

**This image captures the complexities of the disease of patients at the Johns Hopkins Psoriatic Arthritis Program (JHPsAP), who have experienced treatment failure. Each vertical column represents one patient's reported symptoms. Our Patient-Reported Outcomes Measurement Information System-Physical Function (PROMIS PF) Minimal Disease Activity (MDA), a tool developed at Johns Hopkins and validated within our PsA cohort, allows for exquisite description of the phenotypic features of an individual's disease at that moment. Our detailed evaluative approach enables us to match optimal, state-of-the-art drug therapy to the specific clinical circumstances of our patients.**

*Deborah* developed cough, psoriasis and arthritis flare on her TNF inhibitor, which had to be discontinued. We switched to an IL-17 inhibitor and after several doses, Deborah reported diarrhea. She was immediately seen in the Johns Hopkins IBD center, where she had high fecal calprotectin and on colonoscopy, new-onset Crohn's disease was confirmed. We switched to an IL-23 inhibitor and Deborah now has fully controlled psoriatic disease as well as biopsy-proven full remission of her IBD.*

## Multidisciplinary Care

Specialists in Rheumatology, Dermatology, Gastroenterology, and Ophthalmology work together to come up with a consolidated treatment plan. Patients, like Deborah, above, often have problems that can include inflammatory bowel disease (IBD) in addition to psoriasis. Managing this complex disease requires patience, determination, and discernment.

We are very fortunate to have the Musculoskeletal Ultrasound and Injection Clinic (MUSIC), led by rheumatologist Jemima Albayda, M.D. Ultrasound is unparalleled in finding out whether there is true active inflammation in the joints, entheses and tendons versus residual damage from prior arthritis activity. Ultrasound can help at all stages of PsA care: initial diagnosis and during follow-up on established treatment. Dr. Albayda may also find sonographic evidence of subclinical inflammation with joint swelling, and then can take a sample of joint fluid (with ultrasound-guided arthrocentesis) to determine whether there is gout and to quantify the extent of joint inflammation (synovial fluid leukocyte count). Often, this insight guides our treatment approach.

*Brittany* developed extensive cutaneous psoriasis during on and off courses of adalimumab. Her ANA was positive at high titer. However, a skin biopsy confirmed typical psoriasis and was negative on immunofluorescence for immunoglobulin and complement deposition. Brittany had complete clearance of psoriasis and arthritis remission on an IL-17 inhibitor.*

## Why Timing is Critical

About 4 percent of American adults have psoriasis, and of those, one-third are at risk of developing PsA. Because there is no biomarker to predict whether someone with psoriasis is at risk for progressing to PsA, the diagnosis is clinical and time is of the essence. Misdiagnosis can have serious consequences: Within six months, PsA can cause irreversible joint damage. However, people who develop PsA and have access to state-of-the-art treatment, particularly biologics, and commence appropriate treatment within six months are less likely to develop damage and disability in the long term.

## Not Always an Easy Diagnosis

Diagnosing PsA includes a physical examination to determine if the joints and tendon insertions are swollen and tender; a blood test to exclude rheumatoid arthritis (RA); an X-ray of the hands and feet to establish a baseline and to look for radiographic evidence that fits with the pattern of joint injury caused by PsA. Skin disease is a big part of the diagnosis, with psoriasis ranging from a patch in the scalp, or nail psoriasis, or inverse psoriasis, to severe erythroderma. But some patients have no skin disease at all.

Often when we diagnose PsA, it is not acting alone: gout may be affecting the joints above and beyond the underlying PsA. Even if it's "just" PsA and you rule out everything else, the disease itself is complex and heterogeneous, with manifestations including skin and nail psoriasis, inflammatory arthritis, spondyloarthritis, dactylitis, and enthesitis. Ultrasound, again, proves invaluable: it can show areas of hidden inflammation inside the small joints, in the tendon insertion, or around the tendons.

This is why we reassess frequently: every 3 to 6 months. Are we at the target?

## Subsets

Although we have some clinical subsets of PsA, they were established when this disease was initially described, and they describe patterns of articular inflammation. Psoriasis subtype and location add another layer of complexity: scalp, nail and inverse psoriasis are associated with the highest risk for psoriatic arthritis. However, these subsets don't really predict different disease trajectories or individual responses to treatment, and we still don't have a good way to identify patients who share similar disease mechanisms. Enthesitis and dactylitis are associated with more damage. A high disease burden and existing radiographic damage when the patient presents can also be associated with a worse prognosis, and these patients need more aggressive treatment upfront with disease-modifying anti-rheumatic drugs.

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**Dr. Orbai is an Associate Professor of Medicine in the Division of Rheumatology at the Johns Hopkins University School of Medicine. Her clinical focus is PsA and spondyloarthritis.**

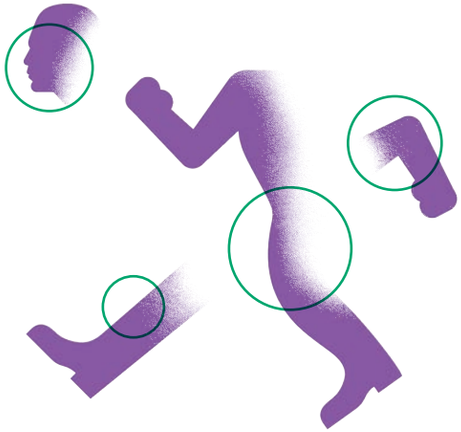
**Internationally recognized for her contributions to the field in the development of patient-reported impact measures and minimal disease activity assessments in PsA, Dr. Orbai has an active clinical research program aimed at understanding psoriatic disease subtypes and improving treatment strategies for people with PsA. She was awarded a Rheumatology Research Foundation Scientist Development Award in 2014, and a Jerome L. Greene Foundation Scholar Award in 2018.**

**Dr. Orbai earned her M.D. from the Iuliu Hatieganu School of Medicine and Pharmacy, Cluj-Napoca, in Romania. She completed internship and residency training in internal medicine at the Johns Hopkins University-Sinai Hospital program, and then completed her postdoctoral fellowship training in rheumatology at Johns Hopkins. During her fellowship, she earned her Master in Health Sciences degree in Clinical Investigation at the Johns Hopkins Bloomberg School of Public Health.**

*Jeff* was bedbound with active bilateral hip arthritis that has been longstanding and refractory, and was at the stage of trying some of the prior biologics at the second round. For this patient, it took a JAK inhibitor combined with temporary anticoagulation to restore his health and functional status.*

## Tailoring Treatment

If someone comes in who already has radiographic evidence of joint damage from PsA, it means they have a pretty severe form of the disease, and we want to get them on very effective treatment without delay. But if symptoms are just beginning and there is no damage, it's reasonable to start with an NSAID. However: Knowing PsA is a disease that is not going away and has the potential to cause damage, follow-up is critical. If control hasn't been achieved, if the symptoms are not gone and if there's persistent joint inflammation, we usually go to a disease-modifying drug that doesn't just take away the symptoms and the swelling, but prevents the immune system from causing further erosive and proliferative damage in the joints, tendons, spine, or wherever the PsA manifests.



We treat our patients in the context of all of the disease manifestations that they experience.

This is why we reassess frequently: every 3 to 6 months. Are we at the target? If we are not, we map out why that is the case, and we tailor treatment accordingly. If we are at target and we continue treatment, hopefully, patients experience long-term remission.

*Kim* has MS and very focal psoriatic disease with nail and DIP involvement that is oligoarticular. She needs a CD-20 inhibitor, which can worsen psoriatic disease. We have been using apremilast, but disease progression identified on ultrasound has made us decide as a team (this time with Neurology) to start an IL-23 inhibitor.*

## Treating the Whole Patient

We treat our patients in the context of all of the disease manifestations that they experience, matching the extent and type of active disease sites to state-of-the-art therapies. We develop an individualized treatment plan informed by the patients' choices and their own definition of a best outcome for themselves. This may involve prioritizing certain disease aspects when treatments lead to imperfect results, while ensuring surveillance and the opportunity to intervene if the risk of damage from the disease increases.

*Louise* has plaque and pustular psoriasis that have been relatively well controlled with an IL-17 inhibitor, but the ultrasound is showing active enthesitis and arthritis supportive of the active symptoms the patient describes herself. We escalated the dose of IL-17 inhibitor to dermatologic loading dose and added apremilast.*

**We have clinical tools to treat-to-target:** Systematic assessment at the clinical visit is part of our treat-to-target (T2T) approach. We use the MDA (minimal disease activity) checklist and the DAPSA continuous score cutoffs for remission and low disease activity.

**So why did we seek a better tool?** Patients who are receiving newer biologic agents are now functioning above average, and early detection of deterioration in clinical status in those with high levels of physical function is essential to providing optimal care. This reflects a shift in focus from preventing disability to the more stringent goal of restoring each individual's best functional status.

Our patients complete the PROMIS PF questionnaire, and then together, we walk through their journey: What is active, and where is the medication failing the patient? Sometimes this can be subtle. In all cases, the goal is to come up with an individualized treatment plan to guide the patients on what their choices are to achieve their best outcome.

*James* has divergent axial symptoms and erythrodermic psoriasis. Combination TNF inhibitor and Il-23 have been required to restore his health and function. Finding the right combinations of biologics truly requires the art and science of medicine.*

*Patients' names have been changed to protect their privacy.

## FURTHER READING

**Measurement of minimal disease activity in psoriatic arthritis using PROMIS-Physical Function or the Health Assessment Questionnaire-Disability Index.**
*Arthritis Care Res (Hoboken)*
2020, PMID:32860727

**International patient and physician consensus on a psoriatic arthritis core outcome set for clinical trials.**
*Ann Rheum Dis*
2017, PMID:27613807

**Ixekizumab for the treatment of patients with active psoriatic arthritis and an inadequate response to tumour necrosis factor inhibitors.**
*Lancet*
2017, PMID:28551073

**Effect of filgotinib on health-related quality of life in active psoriatic arthritis: a randomized phase 2 trial (EQUATOR).**
*Rheumatology (Oxford)*
2019, PMID:31624837

**Effect of Secukinumab on the Different GRAPPA-OMERACT Core Domains in Psoriatic Arthritis: A Pooled Analysis of 2049 Patients.**
*J Rheumatol*
2019, PMID:31615919

**Determinants of Patient-Reported Psoriatic Arthritis Impact of Disease: An Analysis of the Association with Gender in 458 Patients from 14 Countries.**
*Arthritis Care Res (Hoboken)*
2019, PMID:31609525

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**Patients enrolled in the Johns Hopkins PsA Registry since 2016**

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