Transcript Details
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Treatment Approaches for Psoriatic Arthritis

Dr. Neal Bhatia:
Hi, I'm Dr. Neal Bhatia. I'm Chief Medical Editor of Practical Dermatology, and this is another installment of the IL-17 Journal Club. I am proud to have my good friend, Dr. Naiem Issa. Dr. Issa's 3,000 miles away from me, but he is an adjunct assistant professor at both the University of Miami and at George Washington University.

Naiem, you got a nice background there. Looks like you're pretty comfortable. It's good. Staying out of the cold, hopefully.

Dr. Naiem Issa:
It's great to see you, Neal. Thank you so much for having me.

Dr. Neal Bhatia:
Absolutely. No, it's always a joy. Let's talk about an article that was kind of dedicated to psoriatic arthritis. You and I are both proponents of keeping scope of practice high among dermatologists, especially when we don't want to keep giving psoriatic arthritis away to the rheumatologist. I think this is good data for all our dermatology colleagues to talk about, again, some new data, specifically with bimekizumab, but also, again, just talking about the clinical perspectives.

Article is titled "Emerging Targets in Psoriatic Arthritis: Dual IL-17A and F and Inhibition and Clinical Perspective." And I'm just kind of curious, off the top of your head, give me psoriatic arthritis and a synopsis and what dermatologists should know before we get into therapy.

Dr. Naiem Issa:
So essentially, first and foremost, it is really important. I totally agree on having dermatologists keeping psoriatic arthritis, at least in part, in the house of dermatology, but also in collaboration with rheumatologists, because sometimes getting a formal diagnosis may be difficult, and so we need to work in collaboration with rheumatologists. But nonetheless, it is quite important that, as part of our physical exam, that we are not just looking, but we're also touching and feeling.

And when it comes to asking about joint pains, we should be screening our patients at every psoriasis visit. First, identifying potential for PSA or plaque psoriasis, and then further taking that questioning to the joints. Asking about joint pain in general, where that joint pain is, trying to localize that if at all possible. And when is that joint pain happening? For example, is it first thing in the morning? Are we gelling into the day? Are they feeling better throughout the day? Is it more so at night or all throughout the day?

And also poking and prodding. So, for example, you may have multiple joints in the hands, for example, and you would want to touch for potential swollen joints or tender joints or what have you. You don't have to be a rheumatologist to appreciate this. So nonetheless, this questioning can be highly sensitive, along with your physical exam, which can be very brief, to identify a potential PSA patient.

And you may want to then do imaging yourself if you're comfortable doing that or in collaboration with a rheumatologist. But then furthermore, from there, we do have options based on the pathophysiology, which we'll get into momentarily with you and our choice of therapeutics.

Dr. Neal Bhatia:
No, that's perfect, the synopsis there. I think, again, I remember early on in dermatology, people were talking about you can examine a psoriatic arthritis patient just how they walk in the room, just how they use their hands, how they shake hands with you. But to get into detail of about Bouchard's nodes, Heberden's nodes, to get into synovial thickening, that might be a little bit more advanced in some training, but it doesn't have to be.

And it also just requires us to remember that the joints are just as potentially involved as the nails, the scalp, because of memories and everything else in psoriasis. So I think those points are well taken. My biggest concern, though, is we give away, again, the concept and say, "Oh, let the rheumatologist handle it, let everybody else handle it." Will we lose our edge on biologics? Because we're already seeing still not a lot of dermatologists are still writing biologics, not like, what do they call it, the 20/80 rule of that.

Talk to me a little bit about how the biologics patient should be, again, examined, not just head to toe for BSA and PASI, but also, again, what is their rheumatologic assessment in front of the dermatologist?

Dr. Naiem Issa:
Yes. Again, it's a great point. So first, to your point about physical exam, when I am looking at the plaque psoriasis patient at first, I also look at the high impact sites or special sites of interest. So this includes scalp, nails, intertriginous areas. These are regions that we know have a higher preponderance or higher risk for developing psoriatic arthritis. So if I identify a scalp psoriasis patient or a nail psoriasis patient, I'm automatically in that mode of trying to go after the joints.

Now, to your point about managing any possible psoriatic arthritis. Now, I think folks, such as our colleagues, may be a little bit shy of starting to treat early for psoriatic arthritis without having a formal diagnosis. And I think that we need to kind of rethink that. So while we are not rheumatologists and we may not be necessarily the ones to diagnose and pinpoint a fashion of psoriatic arthritis, we can identify joint pain and high likelihood of PSA.

And we do know from the data that early intervention with biologics, and especially as we get into IL-17 world here, will be that you may be able to early on prevent the progression of psoriatic arthritis, while also reducing the inflammation in the joints and the synovium. So early identification of the possible PSA patient and early intervention with biologics here is going to be key.

Dr. Neal Bhatia:
So let's build on that, because that's an important point about where our approach lies on our approach to therapy. And you hit on one of the biggest parts that's in the article. They say 30% of the patients with psoriasis have some element of arthritis or even arthropathy that could be a precursor to arthritis within that.

But the thought that we can approach a more complete attack against arthritis with inhibiting both 17A and F, as well as the other subtypes, if you will, is that the approach that maybe we have to think, "Let's ward that off early on or maybe make that our approach early on," rather than have to go through steps that really don't work?

And even more so, do we maybe think these are automatically biologics patients and not DMARD patients or other oral therapy patients?

Dr. Naiem Issa:
So to the point on early intervention, what we do know is that with psoriatic arthritis is that time is joint, and you can have irreversible joint damage. So that enthesitis or that dactylitis or that axial involvement and so on and so forth. So the longer we wait, you're going to have a harder time of getting to those endpoints of disease clearance, if you will, when it comes to the ACR viewpoints, as well as for that disease progression, radiographic progression of disease. So that's one point.

When it comes to DMARDs, these DMARDs in general are non-specific immune suppressants and/or immunomodulatories. We're talking about methotrexate and the like, and possible Plaquenil, which actually, in my book, I think that looking at hydroxychloroquine, I should say, in my book, it's not true immune modulator in the classic sense. And so I think that we have our targeted approach here with IL-17s.

And you brought up A and F, which is great, but in taking a step back from just those cytokines is the IL-17 milieu, if you will. There's an important concept when it comes to psoriatic arthritis that I think all clinicians, including dermatologists, need to know in general, is that over time, not only do we have the IL-23 pathway, where you have, starting from TNF, goes through IL-23 from the monocytes, and the IL-23 goes and stimulates the T-helper 17 cells to release a slew of IL-17 cytokines A through F, and they go and signal through the receptor on the effector cells, which are immune cells as well as keratinocytes, as well as synovium. We have your synoviocytes, which are very critical in the joints.

At some point, what happens is that you have IL-23 independent IL-17 signaling, and what the heck does that even mean? One, it means that we have an addiction in our immunology for the IL-17 circuitry that feeds forward on itself without the need for 23. The second part to that statement also means that you have a slew of cells, such as gamma-delta T-cells and other innate-like lymphocytes, or ILCs, if you will, that in their own right, which are not T-helper 17 cells, these are cells that can secrete IL-17 on their own.

And we have studies looking at transcriptomics and what have you in the synovium of the different joints that show these cells producing

IL-17 independently of IL-23. So in summary, that leads to the fact that we need to approach this in a targeted fashion, focusing on the IL-17 landscape.

Dr. Neal Bhatia:
Yeah. Well, no, all that makes the case for not only getting after the, if you want to call it master cytokine in this process, you can. I mean, we would debate between 17 and 23 all day, and others have their bias in that respect.