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Patients with Psoriasis: Addressing Advocacy and Education Gaps

Announcer:
You're listening to *On the Frontlines of Psoriasis* on ReachMD. And now here's your host, Dr. Raj Chovatiya.

Dr. Chovatiya:
Welcome to *On the Front Lines of Psoriasis* with ReachMD. I'm Dr. Raj Chovatiya, Clinical Associate Professor at the Rosalind Franklin University of Chicago Medical School and Founder and Director of the Center for Medical Dermatology and Immunology Research in Chicago. And joining me today to talk about education gaps in advocacy and psoriasis is a pretty big deal himself, Dr. Jason Hawkes. Dr. Hawkes is a medical dermatologist in Sacramento, California, and an expert in all things psoriasis and inflammatory.

Dr. Hawkes, welcome to the program.

Dr. Hawkes:
Thanks, Raj. Always a pleasure. Double trouble today.

Dr. Chovatiya:
Well, on that note, to start us off, why do you think there are so many gaps in patient education when it comes to psoriasis?

Dr. Hawkes:
Yeah, I've thought about this a lot, and I think what we have is really an overabundance of information, and I think the medical information that we can give to patients just pails in comparison to the huge amount of volume of data and information thrown at patients online, social media. And I think we get these little, tiny fractions of time with our patients, and then they get these huge amounts of time getting reinforced messages from external, often unvalidated, sources of information, and I think that creates a problem for us as we try to educate patients. We spend so much time undoing misinformation that it's really hard to even get to the foundational information that we'd really like to communicate. And I don't think psoriasis is unique. I think there's a lot of misinformation about a lot of the chronic inflammatory conditions, especially skin conditions, which are so visible, so I think we are fighting a general battle in medicine.

Dr. Chovatiya:
And diving a bit deeper there, and particularly psoriasis, since that's our focus today, can you talk about some of the specific gaps that you're seeing? And maybe, since you've been doing this for a little while, what are some of the gaps, perhaps, that are different now than used to be even 10 or 20 years ago when it comes to thinking about psoriasis?

Dr. Hawkes:
One of the most common things that I encounter with psoriasis patients, and probably even more generically the chronic inflammatory skin diseases, is that there's this question as to what caused my disease, and we see two gaps that fit together in this question that they ask about, when can I stop my therapy? So it highlights, one, that these patients don't really understand that they have a chronic inflammatory disease, but then it also gets back to, "I didn't have it before," so "I'm 38" or "45, and I developed psoriasis as new-onset disease, so if I didn't have it before, then it must not be genetic. There must be some external cause." And so we tend to fight this misconception because we want our patients to start to think the opposite, which is that you have a chronic inflammatory disease. Most of these are idiopathic. We don't really know what sets off these. We know a lot about what's happening when the mature immune response has been established, but what turns it on and off? Mostly on. We rarely see it turn off. That's a hard concept for patients to grasp.

And I think on top of that—especially I want to get to your second question of what's been a new piece of information that's coming out

from patient interactions—is that I think COVID and the immunizations where we saw a lot of things happening, it reinforced this idea that environmental things, such as vaccinations or infections, can sort of turn on diseases, and so there's this concern of could these immunizations or food or pesticides in the fields behind my house, could these be the cause of my psoriasis? We have to go back and avert that where we're talking about these diseases as really being idiopathic in terms of their onset, getting back to this idea of chronic disease. Those are two really common concepts that I see in my interactions with patients.

Dr. Chovatiya:
Yeah. Do you still think that we have gaps here in terms of trying to convince individuals that you're dealing with a systemic-wide inflammatory condition and why we treat the disease the way we do?

Dr. Hawkes:
Yeah. This has been really interesting to watch it evolve, even through my career. So when we started learning about psoriasis, most of the immunology that we learned was from biopsies or blood. So we started to identify these key inflammatory cytokines, like IL-17, IL-23, and even TNF as being markers in the skin biopsy. So of course it made sense that it was an inflammatory skin disease, and then we start to see systemic inflammation as we start to detect some of these same cytokines or signaling pathways in the blood. But what's been a big shift there is we've moved from looking at this as being something in the blood with the immune response for the skin to really being a systemic, chronic inflammatory disease that can manifest in multiple organs. So the skin is just one of the organ systems impacted, but now we're seeing very strong evidence for cardiovascular disease, stroke, hypertension, diabetes, and metabolic syndrome. We're seeing cancer risks elevated. We see joint involvement, so mood disorders, anxiety, depression, and all phases of suicidality. So now we're starting to evolve our understanding to see that the systemic inflammatory environment is driving more than just the skin and joint involvements, which are the two most common.

And that's probably the most common point I try to talk around with patients is that because you have this baseline chronic inflammatory disease, there is an impact, a health impact, of doing nothing. And that's an important conversation piece because we're talking about our patients who say, "I don't know if I want to go on a therapy," whether it's a topical or a systemic therapy like a biologic. They need to understand that there is a risk of doing nothing. And then what we're really balancing is the risk of doing nothing with that chronic, systemic inflammation, which we know has risks, and balancing it then with actually really good safety for most of our systemic medications. And I think that's really been the predominant basis for why we're recommending this wholistic systemic therapy because we're recognizing that just clearing the skin, i.e. with topical medications, doesn't really address the chronic effects and the negative effects of unopposed chronic inflammation in the body, which involves multiple organ systems.

And we're focusing in on the positive aspects of treatment, not only from the skin side, but then saying in addition to the skin and/or the joints, we also have these other health benefits, and I think using positive reinforcement as opposed to fear is important with our patients because there's enough to be afraid about.

And one other aspect that I would mention with the comorbidities of psoriasis that I think is important is the recognition of the baseline risk of a psoriasis patient before treatment, and this is critical when we look at data from clinical trials or research publications. And just because we see a patient, for example, develop inflammatory bowel disease or maybe exhibits suicidal ideation or there's a suicide or we see a cardiovascular event, we have to remember that the control is really the baseline risk of psoriasis without treatment because we know if these patients are not treated, we know they have a high rate of cardiovascular disease; we know that they have a very high rate of suicidal ideation, even suicide or behaviors related to that, and mood disorders, so we see these comorbidities happen all the time. And despite the textbook presentation of skin disease first and then we start to develop comorbidities later if the disease is untreated, we know that that's also not true. We see a lot of patients who will present with inflammatory bowel disease years before they ever develop psoriasis. We see patients who present with psoriatic arthritis manifestations first before they ever develop skin disease. And unfortunately, the typical FDA approach to clinical trials ignores that baseline risk, and so as clinicians, we need to, one, appreciate it, but two, we need to let patients know that it doesn't necessarily mean a drug caused you to get a comorbidity, but your risk was already there.

Dr. Chovatiya:
For those of you just tuning in, you're listening to *On the Front Lines of Psoriasis* on ReachMD. I'm Dr. Raj Chovatiya, and I'm speaking with Dr. Jason Hawkes on the education gaps in psoriasis.

So now that we have a better understanding of some of those gaps that occur, Dr. Hawkes, let's look at some potential ways to overcome them. I think for some individuals it's very difficult to understand whether something is specifically a genetic disease, whether it's an autoimmune disease, the "why" behind why somebody may have psoriasis, why it's a long-term condition. Maybe you can talk a little bit about how you address some of that in really easy-to-understand concepts so people have a better and fair idea of what psoriasis is.

Dr. Hawkes:
Yeah. This idea of a genetic disease I think to the non-medically trained or non-science trained—most of our patients—it's this idea that you have some defect and it causes the disease, but obviously, the genetics is much more complicated than that. I think what's important to note is that, actually, in almost all of the studies that have ever been done, the majority of patients, so more than 50 percent of patients, with psoriasis don't have known genetic susceptibility, loci, and so those are mutations in common genes that are strongly associated with psoriasis. But we have family members within those pedigrees who share the same genetics who never go on to develop psoriasis, and there's variants within even the same gene mutations where we see basically the same type of susceptibility genes in individuals that never go on to develop disease or develop it much later, so I try to talk to patients about things that are modifiers, like a dimmer switch, that it's not the electricity turning on or off a disease, but it tends to be the thing that might turn it up a little bit sooner where you might get an earlier onset or might develop later or certain gene may make are more susceptible for arthritis versus skin disease but that those really aren't definitive and a hundred percent, but they do tend to modify our risk. And I think that's a better way of thinking about it is that the multiple factors that are going to contribute to disease, and it's really the collection or the coordination of multiple factors that are going to drive disease as opposed to one thing.

And, actually, most of our patients we actually never find the gene mutation, and none of those current genes that are associated with psoriasis, for example, are predictive at all for therapy, for example. They don't tell us that this patient is going to respond better to this therapy. So there's a huge gap in our ability to tie the genetic findings to the actual clinical manifestations, and that's been a problem for as long as I've been in training.

Dr. Chovatiya:
As we close out our discussion today in the last few minutes, I'd like to ask if there's any other comments you have in terms of how we can help bridge education gaps, particularly as it relates to advocacy and thinking about diverse patient populations.

Dr. Hawkes:
Yeah. One concern I have, I think, within the specialty is that we're seeing two competing factors. We're seeing less and less practitioners in dermatology who are prescribing systemic or biologic treatments, for example, and I think that is resulting in a worsening access problem for patients, particularly in those more rural areas where there's less providers; maybe they're further away from academic centers. I think, one, we're seeing a decrease in the utility of these agents that we know probably have better efficacy and safety compared to the traditional or historical agents or systemic agents, things like methotrexate, cyclosporine.

And there was a recent publication actually with a Utah group showing that that burden of prior authorizations and the administrative burden related to these types of medications is disproportionate in dermatology, and so that's creating this drive for the finances of business versus stopping, having more time to be able to use these medications effectively to treat our patients. Those are competing interests that are occurring in our specialty, especially with the move with private equity and these big health systems that want to drive high volume because financially it overcomes the cost of running a practice that it's leading to changes in medical practices by individuals in dermatology. And these patients really need better access to these medications, better access to specialists, and I think that's one big area that we need to continue to be aware of and talk about.

And the other area is cost, that luckily, we have a lot of pharmaceutical industry partners that will help support medications to make them affordable for our patients, but there are still these patients that despite healthcare coverage, still can't afford medications that would be medically appropriate or medically ideal for these patients to help reduce the burden of this disease on their overall health. And we need to also keep being involved in that process to advocate for better access, better coverage, lower costs, better support programs where we can help these patients get coverage. And I think that in combination with trying to eliminate the burdens that are deterring practitioners in our specialty from using these highly effective, very safe medications, those two things would have a big impact. And we need to keep those on the forefront in terms of the dialogues that we're having in dermatology.

Dr. Chovatiya:
This has been a great discussion on the education gaps in advocacy and psoriasis, and I want to sincerely thank my guest, Dr. Jason Hawkes, for sharing his insights.

Dr. Hawkes, it was great speaking with you.

Dr. Hawkes:
Raj, thanks for having me. Always a pleasure.

Dr. Chovatiya:
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