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Recognizing Neuroimmune Drivers of Itch in Atopic Dermatitis and Prurigo Nodularis

**Announcer:**
You're listening to *DermConsult* on ReachMD, and this episode is sponsored by Galderma. Here's your host, Dr. Steve Jackson.

**Dr. Jackson:**
Welcome to *DermConsult* on ReachMD. I'm Dr. Steve Jackson, and joining me to explore how disproportionate symptom burden can signal neuroimmune-driven disease in atopic dermatitis and prurigo nodularis is Dr. Diego Dasilva. He's a board-certified dermatologist at Forefront Dermatology as well as an Assistant Professor at Eastern Virginia Medical School in Norfolk. Dr. Dasilva, thanks for being here today.

**Dr. Dasilva:**
Thank you so much for having me; I'm very excited to have this conversation with you.

**Dr. Jackson:**
Let's jump right in, Dr. Dasilva. When you're evaluating patients with atopic dermatitis or prurigo nodularis, what clinical patterns raise your suspicion as something beyond surface inflammation, like neuroimmune signaling, may be driving disease burden?

**Dr. Dasilva:**
So neuroimmune signaling, I believe, plays a role in all atopic dermatitis and prurigo nodularis cases. I think this is something that's evolved in the last five years or even less in dermatology where we thought that there was either inflammatory itch or there was neuropathic itch or conditions in general. And now, we have a deeper understanding that regardless of whether it starts with inflammatory cascades from an impaired skin barrier or an immune system being activated, that will lead to hyperexcitability of a nerve release of neuropeptides that will then cause more T-cell activation, etc. So they feed into one another, and the opposite is true as well. If you have more of what they call a hot nerve or neuropathic condition—which some people put prurigo nodularis in that category—you're going to cause the sensation of itch. Patients are going to scratch, and they're going to then introduce irritants and allergens and stimulate the immune system via the scratching itself. So it's very interesting and convoluted, but it's an exciting time because we're learning more and more about this.

And so when it comes to my evaluation of patients, it really comes down to impact on the patient, right? If their disease is impacting them to a degree where they cannot sleep well or function at work, in social relationships, or at school, etc., then that's a more severe patient for whom I need to escalate treatment. And some patients luckily are not that bothered, and it's either a small surface area or they are living and sleeping fine and maybe can be managed more conservatively with a topical or something along those lines.

**Dr. Jackson:**
Okay, I'm glad you mentioned that. So let's take that a little bit further. Once you start to recognize those signs, how do you characterize pruritus severity in these patients, and what role do associated features, like sleep disturbance, play in understanding overall disease impact?

**Dr. Dasilva:**
A large role. I do like the Peak Pruritus Score—within the last 24 hours, from zero to 10 essentially—because it's something that's used in clinical trials and it's something that we see all the time in papers, etc. So it's a nice way to standardize things. But of course, we all understand human nature, and to attend to someone is not to attend to the other person and vice versa. And so that's where the quality of life impact, plays a huge role for me. I don't spend too much time on this. We have very short efficient visits in dermatology, but to me, that goes with sleep and then activities. So I say, "How much do you sleep?" Number one, right? You ask someone, "Do you sleep

well?" They might say yes, and that's three hours. Well, science shows that's not good for anyone. So I instead ask, "How much do you sleep? Do you feel like itching or your skin disease impacts your sleep at all?"

And then the third piece of that then for me is, "Are there any activities that you enjoy doing or that are a big part of your life—whether that's work or fun or whatever it may be—that are impacted by the itching and skin disease?" And if the answer to any of those questions is yes, then I am willing to be as aggressive with the treatment plan as the patient would like.

**Dr. Jackson:**
And as a follow-up to that, what do excoriation patterns and, in the case of prurigo nodularis, nodular progression tell us about ongoing symptom amplification?

**Dr. Dasilva:**
That tells us a lot actually. So the longer the inflammation is going on underneath the skin, the more lichenification and hypertrophy of the nodules are going to occur and the more nodules you're going to get. But along those lines as well, just the simple scratching of the skin or scratching of an early papule before it becomes a nodule will again stimulate the hypertrophy and the fibrosis to form a nodule. And so these things feed into each other, and unless the cycle's broken, the patient cannot heal.

**Dr. Jackson:**
For those just tuning in, this is *DermConsult* on ReachMD. I'm Dr. Steve Jackson, and I'm speaking with Dr. Diego Dasilva about the clinical patterns associated with disproportionate pruritic burden in atopic dermatitis and prurigo nodularis.

So, Dr. Dasilva, we've been discussing the signals of amplified itch, so now let's move on to how we can assess and act on them. In your experience, how often do you see a disconnect between visible inflammation and what patients report, and what are the limitations of traditional severity scoring systems in capturing that?

**Dr. Dasilva:**
Very often, in fact. I am a pruritus expert in inflammatory disease in my community, so I suppose I see more of these patients than the average person. But even in the past, I've always seen many patients that have a disproportionate burden of itch compared to their skin. And these are patients with atopic dermatitis, prurigo nodularis, or other conditions where the itch can be severe, anywhere from a 7 to a 10. And it could be small areas of surface, like genital, buttock, facial, or something like that where it's not a large part of the body, but it's very debilitating. Or, of course, it can be a large part of the body, right? And the traditional classifications for AD and PN don't help to make the diagnosis or get the patients the treatments that they need.

Let's use atopic derm as an example here. If the genital region is the only part affected, you're not going to be a 10 percent body surface area, even though your itch can be a 10 out of 10 and this can be destroying the individual's quality of life. That makes it difficult then to get an advanced systemic agent approved for this patient. And this is where there's a major gap, I think, in the way that we understand severity in dermatology.

Along those lines, prurigo nodularis is a very classic thing that I see. You might have six prurigo nodules that are well-defined; anybody could identify that level of prurigo nodules, right? But then you may have numerous other spots that are excoriated papules that are just itchy to begin with and there's excoriations. And in my mind, this is essentially a prurigo nodule waiting to happen. It hasn't quite formed itself yet, but it's already active. And so if you go by many of the clinical trial criteria and insurance criteria, they want 20 nodules or greater, and so this patient with six nodules is not technically a candidate, though their disease is severe enough to be so.

**Dr. Jackson:**
With that being said, how has incorporating patient-reported outcomes into your clinical workflow changed the way you identify and monitor high burden disease?

**Dr. Dasilva:**
I think for me, it plays a huge role, and that goes back to what we were talking about earlier: what is the impact on the patient's quality of life? There have been times when I've used DLQI—the Dermatology Life Quality Index. It's a bit clunky and more difficult for me to do in my routine practice. But I will often have a Gestalt for the patient in terms of their itch score and the impact on their sleep or their activities of daily living and enjoyable activities, etc. And so essentially, if the exam is not that impressive—that was what we were just talking about—but if any of the answers to any of those questions are positive and the patient is suffering from that, then automatically they are moderate to severe for my purposes as the clinician.

**Dr. Jackson:**
I have one final question for you, Dr. Dasilva. As we move toward more phenotype-driven care, how can recognizing these neuroimmune-driven patterns influence more individualized therapeutic decision-making?

**Dr. Dasilva:**
I think it's very important to note that it really revolutionizes the entire treatment paradigm. Historically, we would focus on more neuropathic agents or neuroleptic agents, like gabapentinoids, SSRIs, and sedating antihistamines, that could help essentially dull the nerves or make patients sleepy. It was kind of almost a band-aid, right? But now we're understanding that the neuroimmune inflammation plays a role in both.

**Dr. Jackson:**
And with those final thoughts in mind, I want to thank my guest, Dr. Diego Dasilva, for joining me to analyze neuroimmune-driven disease patterns in atopic dermatitis and prurigo nodularis. Dr. Dasilva, it was great having you on the program.

**Dr. Dasilva:**
Thank so much for having me. This was awesome conversation, and I'd just love to see more and more clinicians and folks out there interested in this important topic.

**Announcer:**
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