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Collaborative Consult: Cases, Patient Reflection, and Discussions – What's New in Pediatric Atopic Dermatitis

Announcer:
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Dr. Mancini:
Welcome to this presentation titled: Collaborative Consult, Cases, Patient Reflection, and Discussions. We're going to look today at what's new in atopic dermatitis. I'm Dr. Tony Mancini, I'm a pediatric dermatologist in Chicago at Lurie Children's Hospital, and Northwestern University Feinberg School of Medicine.

Here are my disclosures.

The learning objectives today are going to be to describe the immunopathogenesis of atopic dermatitis. We're going to summarize some current clinical trials data on the safety and efficacy of newer and emerging therapies for this disorder and look at applying evidence-based guideline recommendations for the treatment of patients with pediatric atopic dermatitis.

It's important that we look at these newer therapies and newer observations in terms of the pathophysiology of the disease as we begin to understand the availability of all these new targeted therapies that are now becoming available to those of us who care for these patients. And I want to make a note that the focus of this activity is really pediatric patients with moderate to severe atopic dermatitis.

In any discussion of atopic dermatitis and its treatment, it's really important that we always consider the burden of disease. And this is really significant for patients with atopic dermatitis, and not just a pediatric patient, but their family as well. So, it's important that we always consider things like itch, the itch-scratch cycle, the effects on sleep, the increased incidence of co-sleeping for younger children with moderate to severe disease. And the impact on not just the patient, their schooling, their socialization, but also on the entire family unit, parent work performance, work attendance. There are many factors, which we need to incorporate as we decide which treatments might be most appropriate for that patient.

Mom:
So, in the very beginning, I mean he was a baby, he was - he had one of those dot bands when he was an infant. And I think that was the first time we started realizing that he had some skin sensitivities. It may not have been fully identified at that point. It wasn't until he probably was around the age of like, 1½, 2, he really started breaking out. And that was also when we saw a lot of his environmental allergies getting worse. So, I would say that that really was the trigger.

We started seeing a pediatric dermatologist at that point. And they really started a lot of the topical ointments and such to his skin. And that was really where our starting point began. But I would say 3 was really our turning point where we were like, 'Oh, my gosh, we

have to we have to do something.' Because quite frankly, it was awful watching him go through what he was going through at that time.

I mean, early on, I think it had to have disrupted him a little bit with development-wise. And I say that because, you know, again, thinking back to when he was 3, and I would catch him in corners scratching. And you know, when you have like, toddlers, you know, you know they're being curious, you know that they're exploring. There was a period where, you know, he was young, and I would say that it was consuming.

And when he's had bad instances, you know, when he had bad reactions, you know, it is awful to watch your kid go through it. It was awful walking into a room that the bedsheets would be covered in blood. It was awful to you know, where you're - they're sleep deprived, you're sleep deprived. You're trying to help them at 3 o'clock in the morning, soothe them, but you're feeling awful that your baby, you know, is going through this. You know, and I would say that that was like - those were the worst days.

From a work perspective for us, you know, I don't know that it's necessarily come into so much with work, but there's always that fear. There's always that or a, you know, especially it's no different than when you have a newborn when you're not getting sleep, or either something that's going on.

I mean, this is a disease, this is something medical. And so, it is always on your mind. And you're always worried about the fact that, you know, he's able to be normal and successful and happy and fit in with everybody else. And I would say that it there have been instances in time that he wasn't. But as he's gotten older, and as we've figured out how to use the tools in the toolbox, he's actually started to move into more of that normalcy.

Young Boy:
Yeah, so sometimes I would really want to go to sleepovers, and I couldn't because I had to do medicine, or if they had a dog, because I have - dogs are one of the things that make me itch. So, they had a dog, I couldn't go to their house and have sleepovers.

Mom:
Yeah, I would say that that's a big thing is that you know, what kid doesn't want to go and have a sleepover, go to a birthday party, or go somewhere. And they don't really want mom to come at 8:30, 9 o'clock and say, 'Okay, now it's time for your bath and put on your lotion,' you know.

Dr. Mancini:
So, let's take a minute and look at the immunopathogenesis of atopic dermatitis. And many of you have probably seen these diagrams. This is a very busy slide, but I'm going to walk through it a little bit just to highlight what's going on at the molecular level.

So, if you look at the top of the slide, the epidermal barrier resides here in the epidermis, and it's really important to understand that patients with atopic dermatitis and atopic diathesis have a variety of defects, including a decrease in filaggrin, a really key protein, of which the breakdown products actually help moisturizing and keep the epidermal barrier functioning. Loricrin, involucrin, and really importantly, lipids.

Remember that lipids are a really important vital component of the epidermal barrier. They are the mortar when you think about the epidermis as a bricks-and-mortar model. And the most important lipid is ceramide. And you hear a lot about ceramides or that cera stim with products that are both over the counter and prescription. That's a big part of the disease.

But then let's look down into the dermis. What's going on a molecular level? And you can see here if we look in the middle of this slide, we're looking at the acute stage of atopic dermatitis, you see it's really a TH2 driven process with the cytokine profile you'd expect for a TH2 driven disease. So, interleukin-4, interleukin-13, play a key role. But also, other interleukins, like interleukin-22, which has effects on epidermal hyperplasia and differentiation. Interleukin-31, which really seems to have a predominant association with itch. And then you notice that TH1 does show up here, but it's very small, because it doesn't play as much of a role, at least it's believed in the acute stages, but may play more of a role, if you look over here on the far right, in the chronic phases of atopic dermatitis.

So, it's really a combination of these outside in epidermal barrier defects, as well as the inside out of the inflammatory pathways that combine and contribute to the pathogenesis of this disease. And as many of you will recognize, a lot of these cytokines are newly identified targets for our targeted therapies.

This study was nice in that it looked at the various phenotypes of atopic dermatitis. Many of us were taught in our training that this is a disease that shows up during infancy and goes away during the early school age years. And now we know that while that's true for a lot of patients, it's not true for everyone, and there are multiple different phenotypes.

And if you look at this diagram, it really shows the onset in terms of age of the patient, and when it may or may not go away or resolve.

A few things to highlight. This study found if you look at the curve in yellow, that that early onset, early resolving type was most likely to

be associated with the male sex. At the top in red and light blue, these are the early onset forms, early onset persisting, and early onset late resolving that seems to have more of a genetic association.

And then in purple was a, as of yet, unrecognized subtype of atopic dermatitis, which is a mid-onset resulting type. And in this type, seemed to have less of an association with filaggrin mutations, but a strong association with reactive airways disease.

So, it's important that we understand these pathophysiologic bases for atopic dermatitis as we begin to hear more and more about clinical trials data and newer agents that are being launched and approved by the FDA.

There are some challenges occasionally in the management of atopic dermatitis in children. So sometimes the diagnosis may be in question. It may not be as clear cut as it is in many patients. There is a differential diagnosis for atopic derm. Although to most dermatologists, this is a fairly straightforward diagnosis. There can be features that really overlap though with other conditions.

In terms of treatment, while we mean really don't have the perfectly ideal medication, we have to consider things like the administration route, access to medications, compliance or adherence with therapies, concerns on the parts of patients about allergies or side effect concerns that may or may not be relevant. And there may be a failure to align treatment with guidelines. Remember that we do have many treatment guidelines for atopic dermatitis, but some of them are now becoming a little bit dated and may not include many of these newer agents that are being approved.

There's also occasionally inappropriate use of certain regimens. For instance, systemic corticosteroids are still over utilized. We all know that those are really limited by their long-term toxicity risks, and also by the fact that patients once they go off of systemic steroids, often tend to have rebound flares in their disease.

In one study of treatment patterns, systemic corticosteroids were the most commonly prescribed so-called advanced therapy being used in three out of four patients. And in that study, only about 10% of patients were being prescribed dupilumab, despite it being in the guidelines that are published to date recommended as a next step for moderate to severe disease.

What are limitations of our current options for treatment for moderate to severe atopic dermatitis? Well, on the far left, if you look at systemic immunosuppressants, things like methotrexate, cyclosporine, mycophenolate, or azathioprine, there are a lot of limitations. Treatment is off label for all of these except for systemic corticosteroids. Most of them are really not ideal for long-term use given their long-term risks and potential toxicities. And again, we talked about an over reliance in some areas on the use of systemic corticosteroids.

Phototherapy in the middle. Here, we're talking predominantly about narrowband ultraviolet B phototherapy, which can be very effective, but may be limited by the fact that, at least in children, it's really limited to use in patients 12 years of age, around that age, and older. It can be really difficult for families to get into a dermatology clinic where they have access to light therapy. There are home light boxes available. But those of us in dermatology know that those aren't always as effective. And also, they're not always covered, and they're very expensive. There are issues with cost with travel to sites to receive your light therapy. And remember, this is a controlled form of tanning, so there is a low risk for elevated incidence of cutaneous malignancies, or even eye toxicities.

In the far-right panel, topical corticosteroids overall still standard of care for the treatment of atopic dermatitis, especially mild to moderate disease. But they can be associated with some toxicities and some risks, especially if they're used inappropriately, for instance, too long, too potent, or in the wrong locations. Classic example being a more potent product being used in an occluded area, like the diaper area of a child. Fortunately, atopic dermatitis doesn't occur there, so that's not too relevant to this discussion. But if they're used in other areas where there's increased absorption, like the axillary regions, you could see more systemic risks.

Mom:
Every couple of years, like we get a kind of a handle on it, and then something changes. So, I would say, you know, 3, we got him under control, maybe 5, there was something else that would happen, you know, and he would, you know, he went through a period where he was getting skin infections quite frequently and would be hospitalized for them.

It wasn't until he was probably around 8, 9, I would say, that's really where we started to see more changes. He does more competitive sports. And, you know, the equipment for shin guards or the equipment for baseball or the things that you have to wear, the materials things are a little bit different. He's a boy and wants to participate in that. So, I would say that we've kind of come into ebbs and flows where things get under control, and then it's not under control. And then we have to, you know, regroup, figure out what our game plan is, attack it, and then move forward again.

We have a toolbox here now, you know, that is whenever he has reaction, whenever we have something, you know, we have the tools in our toolbox, depending on the severity. How do we jump in and manage it?

It's hard as a parent because you really have to be dedicated and focused on doing these things. And so, that partnership with the spouse, you know, where there's a lot of communication that goes into play now. It's not just about bills. It's about whether or not you bathe the kid.

He's always had some sort of topical solution with some sort of either cream or ointment depending on the seasonality. Because we found that lotions are better in the summer for things, whereas winter, you know, things like an Aquaphor or something like that, more jelly based, you know, they're sticky, and you don't want that in the summer. So, you kind of are always experimenting to kind of figure out what is going to be working at a certain time. And like I said, things were changing. He's had, what is it the tar wraps, he's had wet wraps, which you could probably tell he wasn't a fan of the wet wraps. You know, I mean, there's been a number of things.

And I would say in the beginning, sometimes it was easier because he is little, and you could make a game out of something. As he's gotten older, it's gotten harder in some ways, because he wants to be responsible for this. He wants to have more control over what goes on and, and he doesn't want to take the time out from the video games or take time out from his friends, or even stop watching a movie to go will take a bath or to go do stuff. And now that he is you know that 4th, 5th grade, it's really important for him, you know, to be clean.

Dr. Mancini:
So, when patients who have moderate to severe disease, it's important to realize that they're seeking care, they're hearing about newer therapies that are evolving. So, it's really important that we are very familiar with the clinical trials data, with the FDA approval process, and with the various agents that are becoming approved for moderate to severe atopic dermatitis. And being openminded to using these new agents is really important for all of us to really help our patients in battling this disease when they're not responding maybe to standard therapies.

So, what are some of the more recently approved therapies for atopic dermatitis? This chart summarizes them as of this point in time. Remember, this is a moving target, and weekly and monthly we're hearing about newer approvals and newer clinical trials.

Crisaborole, a PDE4 inhibitor approved from mild to moderate atopic dermatitis in patients 3 months of age and older. This expanded approval came down in March of 2020. And it had been approved several years prior for patients down to 2 years of age. Really showing promise as a steroid-sparing agent. I think it's still finding its niche in terms of where it fits in. There can be some burning with application, and we have to always consider of course cost and access.

Dupilumab, an inhibitor of both interleukin-4 and interleukin-13 by its inhibitory effect on an interleukin-4 alpha receptor. This is approved for moderate to severe atopic dermatitis and patients 6 months of age and older. That indication down to 6 months of age just came down in the last month or so. In March of 2017, it was approved under 12 years of age with an expanded approval down to 6 years of age in 2021. So, it is administered by injection which may be an important consideration for some patients and families. And again, we have to consider costs and access issues.

Ruxolitinib, a topical agent, which is a JAK1 and JAK2 inhibitor, approved from mild to moderate atopic dermatitis and patients down to the age of 12 years. It was approved to September of last year. Now, if you look at the package label, there is a box warning because the class of JAK inhibitors has a black-box warning. So, there may be a higher risk for serious adverse events. But most feel that with the topical delivery of JAK inhibitors, that risk is far, far lower.

And then the first oral JAK inhibitor approved in children for atopic dermatitis is upadacitinib. This is an oral JAK1 inhibitor approved for moderate to severe disease in patients down to 12 years of age. And it was approved in January of this year for that indication. Again, as I mentioned, as a class, the JAK inhibitors do have a box warning with a higher concern level for a serious risk profile. So that always needs to be addressed with the families, as we discussed with shared decision-making, risk versus benefit. And again, cost and access issues will always be relevant for these newer agents.

Let's look at some of those clinical trials data. And I'm just going to summarize some of the most relevant studies. Here's the pivotal trials data for crisaborole, topical crisaborole, the AD-301 and AD-302 studies. You can see crisaborole in blue and the vehicle in green. And we're looking here at the percent of patients who achieved that goal of an IGA score, that's the Investigators Global Assessment, of 0 or 1. That's clear or almost clear and had a two-grade improvement. Remember, that's a 5-point scale, clear, almost clear, mild, moderate, and severe disease. So, you had to jump to levels or achieve - and achieve a score of 0 to 1. And you can see the crisaborole was statistically superior to vehicle in both of those studies.

Shifting over to dupilumab. This is trials in adolescents 12 to 17 years of age with moderate to severe disease now. On the left, we're looking at patients who achieved a 75% improvement in the EASI score; that's the Eczema Area and Severity Index, one way to capture disease activity in this disease. And you can see the dupilumab in dark blue and light blue at two different dosing regimens compared to

placebo, far statistically superior with a really early separation of these curves. So early onset of action. In the middle of the slide, you see patients who achieved that Investigator Global Assessment score of 0 or 1, again dupilumab in light blue or dark blue, placebo in gray. These are lower numbers, but remember, this is achieving a score of 0 to 1 and you started with a moderate to severe disease.

What about safety, conjunctivitis risk? About 10% in clinical trials. Many of us are seeing less than that in clinical practice. And injection site reactions which are higher than a placebo. But exacerbation of the disease of the eczema and non-hepatic skin infection is actually lower in the dupilumab groups than compared to placebo.

Here's the data in children 6to 12 years of age. So, we're looking here on the far left at the EASI 75. Again, 75% improvement in the EASI score. You see dupilumab at two different dosing regimens in blue, both light and dark blue, and placebo in gray. And you can see it was statistically superior. Remember that in this study, they were allowed to concomitantly use topical corticosteroids. The middle and far right of this slide show when you break this down by weight into those children less than 30 kilograms compared to those on the far right, greater than 30 kilograms. And you see, again, very similar curves. But this was what separated the dosing regimen. So, kids under 30 kilograms, turns out that dosing every 4 weeks was just as effective as every 2 weeks. And that's why that is the dosing regimen for kids 15 to 30 kilograms. It's an every-4-week injection instead of every-2-week injection. Adverse events at the bottom you can see we're very low in both groups.

And here's the trials data presented at the Revolutionizing Atopic Dermatitis meeting in children 6 months to 6 years of age. On the far left, those who achieved an IGA score again of 0 to 1, dupilumab in blue, placebo in gray. You can appreciate the differences there. Dupilumab far superior statistically. And these were all dosed on an every-4-week regimen, and they were allowed to use corticosteroids in both groups. On the far right, those that achieved that EASI 75 score, again dupilumab blue, placebo gray. Dupilumab superior. So being able to use to dupilumab on an every-4-week injection cycle with low potency corticosteroids rapidly and significantly improved atopic dermatitis signs and symptoms. And it was well tolerated. Very favorable safety profiles.

Here we're looking again at the same patient groups 6 months to 6 years of age, same studies, but on the left percent change in the EASI score. And on the right, this is the percent change in the weekly average scores on the numerical rating score for itch. So, the Pruritus Score. And you can see dupilumab in blue on the far right, and placebo in gray. And it was far superior with a really early onset of action in being able to decrease itch. And we now know that itch is such an important part of this disease, that this is really important data.

Moving on to topical ruxolitinib. These are the true AD-1 and AD-2 studies of mild to moderate disease now for this topical agent in patients down to 12 years of age and adults. You can see the first study on the left, the second study on the right, we're looking at ruxolitinib in green, two different strengths, 0.75% in light green, 1.5% in dark green. And it turns out that 1.5% is what went to market. It was statistically superior. But you can see the ruxolitinib compared to placebo was superior in all these parameters, the IGA of 0 to 1, that EASI 75 score, and again dropping in that numerical rating score for itch, at least 4 points or better. Safety profile was good with no new safety signals for this topical agent.

Moving on to an oral JAK inhibitor, this is upadacitinib that I mentioned earlier. And this is looking at patients both 18 to 75 years of age and then also the adolescent trial 12 to 17 years of age. You can see upadacitinib was used at two different strings here, 15 milligrams and 30 milligrams. They were allowed to use concomitant corticosteroids in all groups, including the placebo group. And you can see the upadacitinib in the two orange bars versus placebo in gray. It was statistically superior. The way this was approved was that you start with a 15-milligram dose, you increase to a 30-milligram dose if needed, but you try to use the lowest effective dose. And here on the curve, we're looking at the EASI 75 score. In the middle we're looking at the Validated Investigators Global Assessment score, achieving a 0 or a 1. And on the far right again, looking at an improvement of 4 points or greater on that numerical rating score for itch. It was well tolerated overall. The most frequent treatment emergent adverse events were acne, CPK elevation, and atopic dermatitis flares. I do refer you to the box warning, though, to read up on that, because as we educate patients and parents of our patients about using agents like this, they're going to have those questions if they go home and read that package insert, and that box warning.

So, the availability of these newer agents is really changing the way we manage disease, moderate to severe disease, especially. But even for mild to moderate disease, we now have newer options that are steroid free, not just our calcineurin inhibitors which we've had for several years. And for moderate to severe disease, we really have these new systemic agents, both injectable biologics and oral JAK inhibitors now that are coming into the market. And so, in the past, when we had a patient with moderate to severe disease who wasn't responding to traditional therapies, whether topical, or even light therapy, we were really forced them to consider immune suppressing agents. Now we are practicing in a different era, where we have these newer options that are not immune-suppressing agents, and maybe more effective for these patients with more severe disease.

Mom:
When we began the biologics, and, I would say that that was introduced to me, was right before the pandemic, actually. And he was

starting to have new symptoms. I don't want to say they were new per se, but it was just the flare-ups were different.

We were at a point where they were starting to talk to us about going on, you know, those immune-suppressant drugs and, and the doctor and the dermatologist at the time was like, 'we cannot put him on an immune suppressant drug while they're going into a pandemic.'

I would say within two weeks, we could see a difference. And, you know, it's pretty - it was pretty clear that, you know, things were clearing up. He always has had hot zones. You know, there are certain areas where I, no matter what I would throw at him, like, he always - like his wrist, his elbows, and behind his knees. Those are kind of like the three zones that we always have to worry that, but I can tell you right now if he showed you the front of his legs, he's clear. His back is clear, most of his arm pretty much clear. I mean, it's amazing. And, you know, to start this in September, and kind of saying, like, hey, that I always used to cringe, you know, it's like, wait for that first frost, because when you hit that first frost, then he got clear. Well, he was getting clear before the first frost that particular year, which is great. And he's pretty much remained, like I would say, you know, we've kept it at least 95% controlled, which is very high for him.

Young Boy:
Well, before, I couldn't go to sleepovers, and now I definitely can go to sleepovers and go to people with dogs' house. And I couldn't when I didn't have it. So, I just really like it for that reason. But also, I think, sometimes, like, I would want to climb trees, and the trees would like - like, I'd start scratching, so I couldn't climb the trees. But now because I don't really scratch, I can actually climb trees, because that's probably one of my favorite things to do.

Dr. Mancini:
As clinicians treating these patients, it's important for us to give them information. We're going to talk in a minute about shared decision-making. So, it's important that we share, you know, in a very brief style, the clinical trials data, the indications for the newer agents, the warnings, and the risk-benefit, and really allow patients, their parents and us together to come to a decision about what is the next most logical step for that patient.

Remember that sometimes utilizing these scoring scales in your clinic can really helped justify these agents for insurance coverage. We're going to talk about the scoring instruments in just a minute. I really in favor of the POEM score. That's the Patient Oriented Eczema Measure, P-O-E-M. I utilize that quite frequently in my practice to help document that score, and hopefully help with the approval process for agents like dupilumab, which I've been using a lot of.

So, here's a little summary of several different clinical assessment tools we can use for evaluating the severity of atopic dermatitis. I'm going to start off by saying that most of these tools are used in the clinical research setting, but more and more we've been using them in the clinical setting to help us again hopefully justify approval for the agents.

So, the EASI score we've talked about. It's the Eczema Area Severity Index. The POEM score I just mentioned, Patient Oriented Eczema Measure, the PO-SCORAD. The SCORAD is the Scoring Atopic Dermatitis. The VIGA is the Validated Investigators Global Assessment. And more recently, people have been using something called the IGA x BSA. So that incorporates both the Investigators Global Assessment score, and the Body Surface Area. And you can see all these have established scores and ranges for what qualifies for mild, moderate, or severe disease.

I'm going to focus on the POEM score because it's the one I use most often. You can see 3 to 7 is mild, 8 to 16 would classify as moderate, 17 to 24 is severe, but there's a subset 25 to 28, which classifies as very severe. The POEM score has seven questions, and patients give a response, and can score from 0 to 4 on each of those responses. So, 28 is a maximum score.

So, what about when we use these newer agents? Is it a lifetime commitment? And that's a question that we get asked frequently by families, by other practitioners. And the jury's still out on that. If you notice, package labels don't include endpoints for these treatments. And what I have found is that with some of these newer regimens, they are so effective and lead to such an improvement in quality of life and the patient and their family, that most of them don't want to come off these treatments, I usually suggest at least a 1 to 2-year trial. But as newer agents come on the market, we may find that as clinicians, we start to cycle and maybe try newer agents, maybe there's less frequent dosing in the pediatric population. That's really important. If a patient can inject a biologic every 2 weeks versus every 4 weeks, or if we had one that was every 3 months, wouldn't that be great if it maintained efficacy and safety. So, for right now, the endpoint is not so clear. I think it's really patient specific, clinician specific, and it's something we have to have a conversation about with our patients.

Let's look at some basics of team-based management, shared decision-making, and patient education. So, these are several different strategies which are very effective in the management of this disease. And many of these are true for many disorders that we treat. Multidisciplinary care teams are popping up around the country. They're not available everywhere. But these are units of specialists that

include maybe pediatricians, allergists, dermatologists, nursing staff, psychotherapists, psychologists, counselors, and they really come together for a multidisciplinary clinic experience where patients can maybe see several people at one time and be a part of other maybe support group information. Therapeutic patient education, which is at the bottom of this slide, which is a process whereby we really educate and share management tools with patients, their parents if they're younger kids, and really helped to transfer to them the skills necessary to understand the disease, and really manage it most effectively. And then shared decision-making, which can really improve health outcomes by us offering individualized treatment regimens. And again, educating the families and the patients and letting them be involved in the decisions about what we're using to treat.

Mom:
So, I would say, when you have a kid who has atopic dermatitis, it is imperative that you set up your village. It's really the most imperative part, that you find your trusted professionals, and that they're all on the same page. And so, I would always refer to my doctor's team as his trifecta. And there have been doctors who have come and gone. In the very beginning, the pediatric dermatologist, I felt that she did everything that she possibly could to help him. And it wasn't until we actually had to go and seek out his doctor's team, you know, within the children's hospital nearby, that we were really able, I think, to get the resolve. But it is imperative now that - and it has been through his full treatment, that they're all talking and that they're all on the same page.

We've had to leverage that village a lot, you know, where if he's having a reaction, or if he's having something, we'd have to go to the pediatrician and then they run the test on it. And then it's like, okay, you have to tell his derm, and then his dermatologist can then tell you what to do. It took us a little while to get the team that we have. And I'm glad that we have the team that we do. It's important. And as a parent, I feel supported by them all. And you know, I will tell you that the dermatologist, every single time we see him, he makes a point out of telling me and him that we're doing a good job because this is so very hard to treat and manage. And there are good days and there are bad days. But as a parent, you know ever give up, you know, and it's just always been very appreciated. I've always been appreciative of the fact that he's always, you know, cognizant of like, where our heads are at, and making sure that, you know, we feel supported by him, which is critical.

Dr. Mancini:
So, it's really nice if we can coordinate with our fellow specialists, that's not always possible. But it's important that the patient have their own little village of specialists whom they need and can rely upon to educate them, to follow them when they're starting newer therapies, or when they're using traditional therapies for atopic dermatitis, and to help them when they encounter a barrier and need some modification of their treatment regimen. So, I really believe in this shared decision-making model. I try to practice it with every patient encounter, whenever I can. You can do it quite efficiently in the clinic setting. I realize we don't all have lots of time with every patient visit, but you can get down a few key questions and really cover these grounds in a relatively short period of time.

Remember, our patients are going to have questions, especially in the pediatric world, the parents are going to want to know about safety, safety, safety. How safe is this agent? How safe is it going to be for my child? Is it going to be used long term? What are the risks? What's known about clinical trials? I am honest with families. I tell them that this is a newer agent. We know a lot, we feel very confident in safety overall, but we don't have long-term data. We don't have 10 or 20 years under our belt. That kind of sharing and honesty is really important to help enable them to make the best decision for their loved ones.

So, I'd like to summarize with a few bullet points. Atopic dermatitis is a common disorder that we all manage. And it really can impose a significant burden on patients, on their families. We have to always think about the burden of disease and the impact on quality of life.

Our expanded understanding of disease mechanisms, of treatment targets, and of the different phenotypes of atopic dermatitis really helps us share this with our patients and their families and helps provide us with advanced management options for treating this disease. So, it's a really exciting time to be caring for patients with atopic dermatitis.

For patients who remain symptomatic, despite optimal topical therapy treatment with traditional agents, remember that we have a variety of new therapies. And in the pediatric world right now, we have crisaborole, we have dupilumab, we have topical ruxolitinib, we have oral upadacitinib, all approved for children down to various ages and available for our patients.

Incorporating these newer therapies into our practice requires an assessment of the severity of disease in the patient. I shared with you a few of the different tools you can use to assess severity. I realize many of you probably could do Gestalt rating based on the types of lesions they have, the extensive involvement, and the impact on their quality of life. But really important that we started to document these disease severities with whatever tool you choose to use. And that may also help us get the patient the medications that they need to get access to them with coverage.

Improving multidisciplinary care. If you have access to these multidisciplinary clinics, that's great, utilize them. Shared decision-making with our families and our patients. Therapeutic patient education. These can all really help us provide the most optimal management of

the disease, improve the patient's response to therapy, and really help improve and hopefully maintain their adherence to a variety of topics - of treatments for the disease.

I hope this was really helpful to all of you. Again, as I said, it's an exciting time to be treating patients with atopic dermatitis. While many of us used to dread walking into a room of a patient with moderate to severe disease who was not doing well, now hopefully we all have a new renewed optimism as we walk into those rooms with these newer agents we have to offer and the responses that we're seeing. Thank you very much for your attention.

Announcer:
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