

Transcript Details

This is a transcript of a continuing medical education (CME) activity. Additional media formats for the activity and full activity details (including sponsor and supporter, disclosures, and instructions for claiming credit) are available by visiting:

<https://reachmd.com/programs/cme/experts-master-class-diagnosis-treatment-advances-hidradenitis-suppurativa/56410/>

Released: 04/09/2026

Valid until: 04/08/2027

Time needed to complete: 90 minutes

ReachMD

www.reachmd.com

info@reachmd.com

(866) 423-7849

A Seat at the HS Expert's Table: A Master Class in Diagnosis and Treatment Advances for Hidradenitis Suppurativa

Announcer:

Welcome to CME on ReachMD. This activity titled *A Seat at the HS Experts Table: A Master Class in Diagnosis and Treatment Advances for Hidradenitis Suppurativa* is provided by Clinical Care Options LLC, doing business as Decera Clinical Education, in partnership with the HS Foundation. Prior to beginning the activity, please be sure to review the faculty and commercial support disclosure statements, as well as the learning objectives.

Dr. Peña-Robichaux:

Now, this is the first section of this HS Master Class. So during this first section, what we're going to do is we're going to talk about the pathophysiology of HS to help you guys better understand how this relates to disease progression and also how it relates to our new emerging targeted therapies. We're also going to talk about consensus-driven diagnostic strategies so that we can improve our accuracy in diagnosis in order to start treatment sooner for our patients. Because what do we want to do? We want to help improve patient outcomes.

So starting out with the pathophysiology of HS. Pathophysiology, you know, it's actually very, very complicated, and I think we don't quite understand everything in detail. But at a very, very basic level, what you should understand is this is a genetic autoinflammatory follicular disorder. There is follicular dysfunction that basically leads to a susceptibility to follicular occlusion and rupture, and then that leads to a secondary very robust abnormal inflammatory response. And this response can be exacerbated and triggered by other exogenous factors such as genetic predispositions, hormones, obesity, smoking, mechanical stress and friction, and microorganisms.

But if we're zooming in a little bit more, it's important to know that the inflammatory response involves immunologic mechanisms that lead to broad activation of both innate and adaptive immune cells. With regard to the innate immune cells, there are various cells involved in this inflammatory response, and you can see them listed on the slide there. The adaptive immune response primarily involves these subset of T helper cells, which are listed here: Th17, TH1, and T regulatory cells. It also involves B cells and plasma cells. And this inflammatory response with all of these immune cells leads to an abundance of these proinflammatory cytokines, which include things like IL-1 beta, IL-12 and 23, TNF alpha, and IL-17.

And although there are a lot of cytokines involved in this pathway, the two key cytokines we're going to discuss today, which are central to this inflammatory response, is a TNF alpha and IL-17. And although these both have very distinct immune-cell groups that activate them and by different pathways, they do act synergistically. TNF alpha primarily is secreted by innate immune cells. You see it earlier on in HS lesion development, and TNF alpha also triggers chemokine secretion, including from keratinocytes, fibroblasts, and endothelial cells. This promotes IL-23, which then leads to further Th17 differentiation, which then produces more TNF alpha, which amplifies the inflammation further. When it comes to IL-17, these are primarily secreted by Th17 cells, which are again activated by immune cells. This leads to again production of more cytokines, which then lead to more Th17 differentiation, and again creates this inflammatory response.

With regard to the JAK-STAT pathway, it involves both innate and adaptive immunity, and it's activated by multiple cytokines. When the JAK-STAT pathway is activated, it induces transcription of genes that promote cell proliferation, differentiation, survival, and

inflammation. And one of the key findings as it relates to HS is that there's a strong induction of STAT1 activation in lesional HS skin. In addition, multiple cytokines are involved in HS inflammation signal through this pathway.

And so now that we understand a little bit more about the pathophysiology of HS, let's discuss how we make a diagnosis. So generally, a diagnosis is made clinically. We do a physical exam and take a history. It's important to know that there is consensus-driven diagnostic criteria for HS, and this comes from multiple international expert groups and clinical guidelines. And generally, there's three criteria. Patients must have characteristic lesions in intertriginous areas, typically that recur and are chronic.

These are examples of some characteristic lesions that we see. You can see some typical nodules in the axilla here, some abscess formation which typically means there's a fluid collection that often causes drainage. Another finding you can see in these patients is they're susceptible to forming comedones. And specifically, you can see what are called these double-ended pseudo-comedones, which are almost like these tiny little tunnels that we see. This is important to recognize because you can see patients with earlier disease start to form these, especially in our pediatric population. And then of course the classic tunnels that we see, which can be inflamed and then can also lead to significant scarring, including the specific scarring we see, which is called bridging fibrosis.

So Hurley staging is often used to describe the type and extensivity of the lesions of HS. It's important to keep in mind, though, that Hurley stage is not necessarily correlated with severity. But just to remind you, Hurley stage 1 is typically described as patients who have nodules with very minimal scarring, Hurley stage 2 is when you have tunneling with some limited scarring, and stage 3 is where you have extensive tunneling and extensive scarring. But as I discussed earlier, it's important to remember that Hurley stage does not equate to severity. For example, you can have patients with Hurley stage 1 who have an exuberant amount of nodules and abscesses without any tunnels. HS can present with heterogeneous phenotypes, we all know; we've seen these patients before, they can look very different. And there are many groups that have tried to break these clinical phenotypes down. Personally, I find the phenotypes from the group from Martorell to be most helpful. They've simplified this phenotype, and I have some pictures here of what they describe. The Martorell group describes two phenotypes: inflammatory subtype and follicular subtype. Inflammatory subtype typically is described as what we think of as your typical HS—those inflamed nodules and tunnels that tend to drain. And then the follicular subtype are patients that you see more of a nodulocystic type of presentation. You see more comedones and more of these kind of nodules that you see. Some studies also indicate that when patients present with this inflammatory phenotype, they're more likely to have severe disease and more likely to progress.

It's also important to recognize the risk factors associated with HS. These include things like sex and race. We do know, for example, that Black women are more likely to have HS. There is also genetic predisposition for patients. About 30% of patients have a familial variant of HS, which is associated with a monogenic mutation. Smoking is something also related that we see as a risk factor to HS. It's important to understand that although smoking is not a cause, it can be a trigger, and studies have shown that patients who smoke are actually two times less likely to respond to treatment. Obesity is a risk factor that's discussed a lot. And again, all those studies have shown that patients who are obese tend to have more severe disease. Patients who with normal BMIs can have severe disease as well. So again, very important to tell our patients this is not a causal relationship. And finally, we do know that lower socioeconomic status is associated with HS as well, but this can be a bidirectional relationship.

And so putting this all together, you know, understanding the risk factors of developing HS and how to clinically recognize this condition is key to starting timely treatment. And this is important because delays in diagnosis can lead to things like progression of disease, more severe scarring, worsening comorbidities, more surgical interventions that may not be needed, more of a psychological burden. Patients often that are misdiagnosed will get treatments that they don't need, and this again will cause healthcare costs to skyrocket.

And so keeping this in mind, let's now listen to this patient discuss his HS journey. Unfortunately, stories like this are not uncommon, but hopefully we can change that.

Mark:

Hello. My name is Mark. I am 39 years old. I live in California, and I'm here to tell my HS journey. I've had HS since I was, you know, around 12 or 13 years old. It definitely started around the onset of puberty, and I just treated it and thought it was acne. But, you know, as I continued to get older, the condition and the severity of it continued to increase. And you know, the symptoms were quite noticeable and painful.

So during, you know, my teenage years, I would see my doctor and, you know, started with the primary care and really was just trying to understand maybe what was going on. And, you know, the doctor understandably would look at it and, you know, look at me and, you know, treat it as though it was teenage acne.

And so for many years, that's what I thought. And I would go to the office and see the doctor, and he would prescribe antibiotics and topical washes and lotions and creams and all sorts of things. And they never really ever seemed to work. You know, I would go back, a

different one would be prescribed, and it was just sort of an endless cycle of antibiotics, washes, creams, you know, sort of rinse and repeat. And I was always told, you know, I would grow out of it, you know, it was just normal teenage acne. And so that's how I really lived my life all through my teenage years.

And, you know, into my 20s, I started realizing, you know, the acne that I had had that was on my face and really more traditional places had really sort of cleared up, and I wasn't dealing with the issues. But you know, the flareups and the things I was having on my body never really went away. And if anything, they were getting worse.

And so into my 20s, many of the areas had become quite severe and persistent. And so I had to really pursue a more extreme level of care. I started seeing, you know, dermatologists. I started seeing specialists in everything from, you know, allergy to infectious disease to, you know, immunology and anything I could, you know, try that was a new avenue to try and figure out why I was getting these incredibly painful, you know, long-lasting sort of deep flareup-type infections, and they were constant. I would get new ones almost every single day, it seemed like; there was just not a day where I didn't have a new area that was flaring up.

And so as I continued to try and find treatments, I had to start having surgeries and procedures to remove really constantly infected areas. And one area in particular was on my buttock, and after a number of procedures, the wound was quite severe and required packing and wound care, and it was something that I couldn't manage because of just where it was located. And so a friend of our family who was a nurse was helping me kind of on her side time to treat and care for me. And as she saw what I was going through and she saw, you know, sort of what the condition was, she started asking, you know, around in her circle of friends and, you know, medical network that she knew. And one day, she came back and asked if I had ever heard of HS. Through all my appointments, through all the doctor visits, that condition had never been brought up. She had printed out something from online. It was sort of a description of what the condition was. And as I read through it, it, I mean, sounded exactly like what I was experiencing, you know, flareups on the trunk of my body, under my arms, you know, you know, consistent, you know, sinus tracking, the whole sort of all the hallmark traits of HS were finally described in one place in a diagnosis. And at that point, I was convinced. I knew I had it.

So as I took that information and started doing my own research, I found a doctor who had an HS clinic. And when I saw the doctor, he confirmed almost immediately that it was HS, and that was really where the journey to treatment started with effective treatments.

Over the last 10 years, I have been on three different biologics. Still have the condition. I still get flareups, you know, I still have to go in and get the steroid injections or, you know, sometimes have a drainage. But for the most part, my quality of life and the severity of the condition has improved dramatically.

Dr. Peña-Robichaux:

Okay, so we're going to move on to the interactive part of this session now. What we're going to do is I'm going to present a patient case and then present you with some questions for discussion, and then we're going to break out into some small groups and kind of have discussion around the table.

So this case is actually a personal patient of mine. I saw them a year or 2 outside of residency, and she was a 50-year-old female Hispanic woman. She had over 20 years of history of very, very severe symptoms. She had these chronic and recurrent flares, primarily on her pannus, mons pubis, labial area. And she had been to the ED multiple times and had tried multiple topical medications, multiple antibiotics. And when I met her and we started discussing treatment with biologics, she was very hesitant because she was worried because she's been told this whole time she had cellulitis, which is an infection. How possibly could she start a biologic?

So for this case, what we would like to discuss are these questions. So what features of the history support the diagnosis of HS specifically to this case? In what ways do you think this patient has been impacted by her untreated HS? How does timely diagnosis play a role in the clinical course for patients like this? And based on your understanding of HS pathophysiology, how would you approach clinical management?

So we're going to take about 5 minutes to have discussion.

Dr. Peña-Robichaux:

You guys want to talk about the case?

Dr. Horissian:

Okay everybody, I do see one question that's been asked by you all that I can explain as we're going. So the question was, can you explain Hurley stage 1B/C? And if this is helpful clinically for insurance approvals of systemic or choice of antibiotics and treatment or choice of treatment in general.

I personally don't use the modified Hurley scoring system. I think it's just a little bit more cumbersome, although I do think it's better than the 1, 2, 3. But as far as insurance goes, I find that they're looking for the word moderate or severe, and so I'll usually add that to my

note and the descriptions of what they're going through and how it impacts their life. And I haven't had an issue with approval in that regard.

Does anybody want to message in, you know, any of the answers to the questions here about how timely diagnosis plays a role in the clinical course or how you would approach clinical management of this patient?

You know, I think the recurring nature, the location of the spots all support the diagnosis. I think the impact of untreated disease can be multifocal. One, it can be not understanding your disease, not understanding what causes it, having the misdiagnosis of recurrent infections, and that leads to, you know, inappropriate treatment or not seeking the right person for treatment like a dermatologist or an HS specialist.

I think timely diagnosis plays a huge role in the management of HS because once it gets to a point where there's a lot of scarring and tunneling, it's really hard to reverse that. It's impossible to reverse that in my opinion. And so the earlier we can start people in appropriate treatments, the earlier we can stop that cataclysmic cascade of inflammation and scarring and kind of prevent the disease from worsening, really disease modifying in a sense. So that's how I think the earlier the better.

And that's multifocal because the primary care physicians and the OB/GYNs and the surgeons need to be able to identify what it is and send it to the right people. Otherwise, there's the delay in care of 8 to 10 years that we've talked about in prior studies.

And then, you know, I think based on pathophysiology, how would I approach the clinical management of this patient? And I think based on that photograph on the last slide, has pretty significant, you know, inflammatory disease, and so we need to treat that with something that's anti-inflammatory. And to the patient's question of, if I've had recurrent cellulitis, won't this make me worse? That's a valid

question, a valid point. But because HS is an inflammatory disorder, we use the anti-inflammatory agents, biologic agents, those kind of things, to quell the overactive immune system at the level of the hair follicle and kind of prevent the disease from having as frequent flareups or having as much inflammation there, as much drainage, or having spots happen as often.

Does anybody else have any questions on diagnosis?

We've got a question for the virtual group, and you can pose your answers back as a question to me, but how do you diagnose HS? What's your criteria? And do you find that it's usually diagnosed by the time it comes to your door, or you're the first one making that diagnosis? I know in my experience, I have a lot of patients who are actually diagnosing themselves because their primary care doctors told them it was just an infection, but then they go on TikTok and find that out. So it's one thing that social media has been positive for, for my patients.

A couple of comments here. I see the psychological impact of recurring episodes is huge. Yes, I absolutely, absolutely agree with that.

Okay, this is a good one. So in clinical practice, what is the average time to diagnosis for HS patients, and what would be the comorbidities that matter the most when planning treatment? Great question. You know, I think it really depends on when you catch the patient. If you look at an average pool of all patients, the delay is still pretty similar of 8 years. I find a lot of my own patients are getting diagnosed earlier, whether that's because we've done a lot of work teaching the primary care doctors in the office and the pediatricians or, like I mentioned, social media. So I feel like that's going down. But if you look nationwide, that's not necessarily going down for people.

Comorbidities, I think obesity, smoking, PCOS, you know, the metabolic-syndrome things, those are things we should be aware of because some of our therapies can help more than one thing. I find a lot of these patients aren't necessarily seeing their primary care doctors, and so I kind of function as one. But I think plugging them back in with primary care can be helpful to address those things.

What is the impact of inflammation on other body systems? Another great question. I think that similar to psoriasis, you know, we usually think things are skin deep until we find out there's more to it. So similar to how psoriasis we found out was a systemic inflammatory disease, the same thing is true with HS. And I think people can have other inflammatory conditions like inflammatory bowel disease, ankylosing spondylitis, or even just inflammatory joint disease. A lot of these patients, particularly those with severe disease, have leukocytosis and anemia of chronic disease. So I think the impact is definitely there on other systems.

Another good question. So other than Hurley, what else have you used for diagnosing/determining severity? So I actually base the severity not off my own impression of the patient, but off of how they describe their disease. So I try to ask questions like, is this keeping you from going to work? How many days did you have to call off work in the last 3 months? Is this keeping you from going and doing what you want, like going to the restaurant or the gym or having sexual intercourse? And you know, if there are things that it's changing their life in a negative way, I think that's at least moderate severity, if not more. If they have one flareup a year, doesn't bother them at all, and it comes and goes, and they're not bothered by it, they'd be okay taking antibiotics for a couple weeks when that happens, I think

that's mild disease. But I think a lot of people fall into the moderate-to-severe category.

And I think that if we base it solely off of what we're seeing, sometimes we can misjudge patients, and that's for multiple reasons. We have our own biases. But I also think that HS is a disease that changes day by day, so we don't always see them in their worst moment. They may have flared up a week ago and not the week that we have it. So I use a lot of patient-reported outcomes kind of to determine how much this is affecting them and in turn putting their severity in my notes.

Dr. Peña-Robichaux:

Okay.

Dr. Horissian:

All right, thanks everyone.

Dr. Peña-Robichaux:

Yeah, yeah. All right, so let's just recap what different discussion points we were talking about. I know some of the tables I was discussing, we were talking about how it can be difficult to have that discussion with patients about what they've been told in the past versus what we're telling them at the visit. So for patients who've been told they've had an infection, they have cellulitis, that's always a difficult conversation and to get them to understand that that actually, no, this is something different and that that can be challenging. And what some people were saying is, you know, just building a relationship with a patient to gain their trust and also interacting with other providers, you know, primary care, so that everyone's on the same page with the diagnosis.

Did you guys have any good discussions?

Dr. Barnes:

Yeah, I think at one of our tables, we were talking about, you know, that buy in, that shared decision-making, and with such severe disease and with her maybe being more concerned about it being an infection and being more open to antibiotics, even considering like an IV ertapenem bridge to surgery or bridge to another biologic medication. So, yeah.

Dr. Lee:

Yeah. And then there were some kind of diagnostic challenges that were discussed. So what came up is, how do we differentiate acne versus HS? In all honesty, some patients have both, right? We know acne and HS are kind of comorbid conditions. But I think with HS, a lot of it is listening to that history of the chronicity, looking, you know, and for all of my patients with severe acne, I usually get them into a gown and check them head to toe because you might find HS. So looking kind of behind the ears and those intertriginous areas. And looking for subtle clues because we know that HS just kind of waxes and wanes. So they might be coming to you on a day maybe where their HS is quiet and controlled and looking for subtle clues. Is there any atrophic scarring in that intertriginous area? Do we see those double-headed comedones? And things like that can be very helpful.

Dr. Peña-Robichaux:

And this patient, I still see her to this day. And just to update you guys, I gained her trust. We got her on adalimumab, and she got somewhat better, but then we transitioned her to infliximab, and then I got her to surgery. We removed all those areas that you saw in the photograph, and she is completely clear. She continues to be on infliximab to this day. And that photograph, I believe, was probably from 8 or 9 years ago. She actually had a lapse in insurance and came off her infliximab for a bit and started having a little bit of recurrence. So we went back and did surgery again, and now she's clear. So she's doing really, really great.

Dr. Lee:

Okay, we're going to move on to the next section, and I'll get us started here. So the next section is patient-centered and long-term management strategies in HS. And the goal of this section is to really help provide you with tools to help you formulate comprehensive care plans that take into account patients' comorbidities and their quality of life.

So we're going to jump right into comorbidities. We know that HS, as we discussed in the prior section, is a chronic inflammatory condition of the skin. But if we stop there, we're really underestimating its burden. Patients with HS have not only severe tunneling, sometimes severe inflammation of their skin, lots of drainage and pain, but they also face high levels of systemic inflammation and high comorbidity burden. So it's not uncommon for a patient to have concomitant cardiovascular, endocrine, metabolic, gastrointestinal, MSK, or even psychological comorbidities.

And so I've actually included a QR code here in the upper right corner. This is a nice comprehensive guideline on evidence-based comorbidity screenings that were put together by the US and Canadian HS Foundations. But screening and recognizing comorbidities remains really central to delivering optimal care.

So diving specifically into cardiovascular and metabolic screening, patients with HS have increased risks of hypertension, dyslipidemia,

diabetes, metabolic syndrome, and for female patients, polycystic ovarian syndrome. And as a dermatology provider or our dermatologist providers out there, many of us are not necessarily checking a blood pressure or a lipid panel, and definitely not doing ultrasounds of the ovaries. So this is where it can be really helpful to partner with your patient's primary care provider to help screen for comorbidities.

And at my institution, what we actually do is we provide the patient with a handout that has a list of all of the comorbidities that I would like their primary care provider to screen them for. And so they'll take that over to their primary care provider so they can get that comprehensive screening.

And in regards to inflammatory comorbidities, epidemiologic studies suggest a two- to three-times-higher risk of inflammatory bowel disease, and many patients may also suffer with joint pains. I don't know, you know, if you've asked your patients, many of them tell you they have joint pains. And a lot of those patients may have pains from an inflammatory arthritis. Some of these patients may come into your clinic already having been diagnosed with IBD or inflammatory arthritis. But if they haven't, it's important to screen your patients and ask those questions about abdominal symptoms, GI symptoms, and joint pains.

And what's really helpful is if you have a patient that has a comorbidity like inflammatory bowel disease or inflammatory arthritis, you can then prioritize targeted therapies to kind of target both of those disease states. And so for example, if I have a patient that's coming in with a history of Crohn's and HS, I may be trying to reach for that TNF alpha or even off-label upadacitinib to kind of target both of their comorbidities.

On the flip side, we also want to think about kind of comorbidities in the sense of kind of what prohibits from using or kind of we should caution using certain biologics. So for example, if a patient has heart failure, I might try to kind of prioritize IL-17. Or if a patient has a history of lupus, I might try to steer away from the TNF alphas and prioritize an IL-17.

And research has shown that people living with HS are at significantly higher rates of depression and anxiety. And actually, the literature suggests that this risk stands true across all severities and stages of HS. So what can be really helpful is screening your patients with a Patient Health Questionnaire-2, which is a quick tool. It takes about 1 minute to complete, and it's scored from 0 to 6. If your patient has a score of 3 or higher, you can then either follow up with a PHQ-9 or refer off to a mental healthcare professional for further workup and treatment if needed. And this slide's really just here to remind us that optimal outcomes in HS often depend on a multidisciplinary approach. And as dermatology providers, we really can make an impact in improving patients' skin and quality of life. But when we partner with other specialties, we really can have kind of things optimized to deliver truly comprehensive care.

And then just a note on lifestyle modifications, which many of your patients may ask you about, it's helpful to remind them to use gentle and fragrance-free products. Many of them at baseline have very sensitive skin, especially if they're having a lot of drainage or open wounds. Loose and breathable clothing like 100% natural fibers such as cotton, bamboo, and linen are generally better tolerated. If your patient can't do rigorous exercise because they're in so much pain and they're having a lot of drainage, you can recommend alternatives like swimming or yoga.

And then I also list healthy weight management and smoking reduction and cessation here, which are definitely important for overall health and may help, you know, improve HS symptoms. But I put an asterisk because it's important to really wait to discuss those sensitive topics until you've really built that rapport with your patient. A lot of patients have been told that, you know, their weight and their smoking is the sole cause of their HS, and we know that just isn't the case. So really building that rapport before you dive into some of those more sensitive topics.

And I'm going to pass the mic on to Dr. Barnes.

Dr. Barnes:

Thank you so much, Dr. Lee. So now we're going to transition to quality of life and barriers to care for HS.

And so I know when a patient walks in the room, we really are just seeing the tip of the iceberg. We may see some painful nodules, abscesses, some scarring and some sinus tracts or tunnels, which is the better term that we use now, and drainage and odor. But what's really lying beneath and what's really driving a lot of the suffering is the chronic pain, the depression and anxiety, the social isolation that's also related to stigma and embarrassment that truly impacts all relationships, both work and personal.

And HS actually has some of the highest scores for burden and quality of life. So DLQI, which is the Dermatology Life Quality Index, some of the scores for HS, as you can see in this image to the left, are so much higher than other conditions that we treat, even urticaria, which we know can be very itchy and keep people up at night, and atopic dermatitis and psoriasis. These all are compounded by the barriers that our patients are experiencing. And so this is a large part of the research that I do. And in a study that I did with one of my mentors, Dr. Haley Naik at UCSF, we identified structural barriers, healthcare-provider barriers, and also patient barriers. And so as

you can imagine, for anyone from an international like health system, top one probably doesn't apply as much to you, but health insurance coverage in the US is really difficult. And for a lot of our patients, especially those from lower socioeconomic status, this drives a lot of barriers to even accessing a dermatologist and accessing the FDA-approved medications that we know can really help them.

There are costs—not just the costs of paying for medications, but wound care, the costs of missing work because you live 4 hours away from an HS specialist or a dermatologist. So just thinking about all the ways that these can be compounded.

And with the healthcare-provider barriers, I do a lot of qualitative work as well. And some of the things that unfortunately we've heard that some of our colleagues have mentioned to patients or, you know, sighs of disgust when they see their HS or, you know, just not even wanting to touch or examine, that really gets internalized and makes people want to avoid health systems altogether. So our attitudes can play a big role in breaking down some of those barriers for our patients.

And I know we don't have a ton of time, but it's really important to try to address a lot of these barriers. And I previously had a QR code that took you directly to the HS Foundation website. But if you can Google HS Foundation prior authorization template, they've already done a ton of work to make accessing these medications so much easier for patients. You can use these in terms of prior authorization, so almost attaching a letter of medical necessity to the bottom of your note. But a lot of times what dictates what gets covered is if the patient actually has moderate or severe disease. And our notes, if they're not detailed enough—describe the areas of involvement, describe the suffering that patients have, missing work, needing to be on disability—then that is almost a disservice in getting that medication for our patients.

Outside of just what's covered by insurance, there are some other financial assistance programs that a lot of our industry partners have in place. So there are patient assistance programs. There are also free drug programs, and linking your patients with those or offering, you know, someone in the clinic like an MA can look things up with them and just see, okay, we know your insurance didn't cover this, but here's another option to make sure that it's accessible.

And if you do unfortunately get that denial, please do the peer-to-peer. There are so many times where I think it's almost an automatic denial at times, and all you have to do is hop on the phone and advocate and say like, I'm a dermatologist. I take care of patients all the time, and this patient has some of the worst HS I've ever seen in my life, and this will truly improve their life.

And then there's also support navigation just outside of seeing you in clinic. Sometimes it's hard to coordinate with primary care and with you and with surgery. And so if there are models that your health system allows, like if there's social work that can help patients navigate this, that's really helpful.

And then also addressing barriers. Again, there are so many social determinants of health or drivers of health that are before a patient even comes into the visit. So even asking about food, transportation, housing. Some of our patients are trying to decide between buying groceries and buying that medication. And if we never ask, then a lot of times they don't volunteer.

And so to quickly incorporate some of these things that we can help to improve the quality of life, just listening and being empathic as best as we can, especially being gentle with physical exams. So this is just a picture from my clinic in Emeryville in the Bay Area. It's really unfortunate if you're draining and you sit down on that really thin paper that's going to stick. So trying to make sure that there's a chuck so that patients feel a little bit more comfortable if there's draining, that they're not going to have that uncomfortable movement when they're rotating for you. A pillow—this is a painful condition, so providing that just allows someone to adjust the weight as they need. And then I also always provide an extra blanket, especially if someone's a little bit on the larger side and the gown isn't covering as much, it helps provide some sense of modesty if they're able to use an extra blanket to drape.

Always assess the areas of involvement, count lesions if you can. This is all important for advocating in your documentation. Multidisciplinary care, as we've discussed, is important. And then support groups. So there are so many support groups online for our patients to connect with, even outside of the visit, and they can be a real source of strength and resources as well.

And then last—this is a busy slide that you don't need to digest—but pain. Pain is one of the most impactful symptoms of HS, so please ask about it. Please try to address it. You know, first line would be over-the-counter Tylenol and NSAIDs like ibuprofen and naproxen sodium, but there is a ladder that you can go through. And so please feel free to use this resource. And there's also a wonderful article that summarizes these as well.

And with that, I'll take it to our case. We have a 35-year-old woman who presents with recurring and tender bumps in the groin, inner thighs, and axilla every 1 to 2 months for 6 years. One area is raised and drains from two different openings. She reports that she's missing work due to pain. She lives with her husband and her two children who are young and have a lot of needs. And her uncle has always had boils, aka there is a family history of HS. The past medical history, she also has prediabetes, PCOS, acne, and she notes that her HS is a little bit worse or she has acne flares around her jawline, and she also has flares around periods and reports some

depression. She's currently on metformin for her prediabetes and tretinoin for her acne, and she doesn't have any known drug allergies.

So now we'll just like to take, you know, a few moments to discuss this case. Especially since HS is frequently associated with systemic diseases, what additional comorbidities would you screen for? How might her PCOS and prediabetes play a role? And what additional questions would you like to ask her to see how HS is impacting her life? How might you educate and empower her with self-care strategies? And what social determinants of health may influence her treatment decisions? And what resources and interventions could you help provide to address these barriers?

And with that, we'll go out into the group and discuss.

Dr. Horissian:

Okay, thanks. Okay, so virtual audience, what do you guys think? We've got a couple questions up here on the screen. What else should we screen for? How might PCOS and prediabetes influence your treatment management?

Personally, it would make me think about if she wasn't on metformin, potentially starting her on metformin to both help with the prediabetes and the HS. Since she's already on metformin, we could consider going up on that dose for the prediabetes. That's one option. PCOS would make me think, is she having flareups with cycles or are the cycles kind of irregular? Might she benefit from spironolactone, which could help with some of the symptoms of PCOS and hirsutism? Depending on the age and other comorbidities, are oral contraceptives a good option for her? So there's a lot of different options based on the comorbidities that we could think about in addition to biologics.

What kind of questions would you ask to better understand how it's affecting her life? Personally, I usually ask them just point blank, how is this affecting your life? And then they sometimes break down in tears and tell me, you know, 'I haven't been able to go to work,' or 'haven't been able to be with my partner,' things like that.

There's a good question here. I'm just reading it. So two questions. Do you find certain drug classes work better for certain morphology types? For example, IL-17 over TNF for draining tunnels? Potentially, yes, I do think so. I think we need more data on that to really prove it so it's not just anecdotal evidence. But I do think that draining tunnels might be more helped by IL-17 inhibition. The second question is, Hurley is a flawed system, especially for assessing response. Do you use a different scale like the HS-PGA or IHS4 in clinic? I know several people do use different systems. I usually don't unless the insurance comes back and tells me I need a specific measure. I think those are really helpful measures, particularly in clinical trials, but in a busy dermatology clinic, you know, counting every single abscess or nodule, not always practical. So I generally just base it off of what the patient tells me and clinical photographs. So I take clinical photographs at every visit and kind of compare. Again, those are just a snapshot in time. And to your point, you don't go backwards in Hurley staging. So it's not a great system other than, you know, giving it a number and getting insurance approval with that. So I usually base it off of, do I feel like they're having less inflammatory lesions? Are they having less frequent flareups? How many flareups do they have a month? And those kind of questions let me know if they're really getting better or not.

Great cases and pearls. Can you talk a little bit about sweating? I often think about Botox in my more mild patients, but maybe I should be thinking in severe patients too. So I agree. I think there are some studies that show that Botox can help. And with the sweating, follicular occlusion, the moisture, the rubbing, I definitely think it can play a role in some patients. I don't think it's going to reverse somebody that's stage 3. So I think if it really is helping with the disease, it would probably be more helpful earlier on in disease but could be helpful in later stage if they have excessive sweating.

And then maybe you will get to this, but can you talk about goal dose of metformin for HS? Yeah, we don't have that specifically in the talk, so I'm happy to answer that. I think it depends on tolerability. So I've seen people on a pretty high dose of metformin for their diabetes. When I usually do it, I usually do 500 once a day up to twice a day. I find two pills a day, so 1,000 mg a day, tends to be where most people balance out from a side effect profile standpoint. When I've gone above that, they've usually gotten sick from it. And even some people that get two pills a day just doesn't work well, and so we do one 500-mg dose per day.

Great questions. Do you guys have any other questions out there?

Okay, I see one here. So talking about a patient that has a history of a recurrent solitary nodule of the groin and buttock and one scar on the buttocks, do you suspect this is HS, which aligns with the possible underlying ovulatory dysfunction that you're evaluating? Yes, I agree. You know, usually when you have recurrent lesions in the common hot spots and they keep coming back and draining or even scarring, that's usually HS. So I agree with you on that. Are there any social determinants of health that influence, you know, treatment decisions or any resources or interventions that can help you or your patients?

Dr. Barnes:

This is pretty fun.

Okay, and so great. Let's go ahead and do a recap. We heard some really great discussions amongst the tables. People picking up on the fact that she has PCOS, she has acne with perimenstrual flares. Spironolactone would be a great way to address that. Also making sure that we screen for other comorbidities like maybe some arthritis or also IBD would be really important as well.

Dr. Lee:

Yeah, PCOS, I think a good alternative or you know, if some patients that are on spironolactone and birth control, and that came up in discussion, I think what's important to remember is not all birth controls are helpful for HS, and some can actually worsen symptoms. So when you look for birth control, we really want to look for one that has that newer-generation progesterone, so that fourth-generation progesterone, generally like a drospirenone that has antiandrogen properties. So if you, you know, are looking for birth control, that you would kind of try to look for a newer-generation progesterone.

And I think GLP-1s came up in the questions. And a lot of questions came up on GLP-1s. I think it's a very hot topic in general, and it's shown some benefit for a few disease states like psoriasis, you know, even HS, chronic wounds. Right now, we just have very preliminary data, but hopefully, you know, hopefully we have kind of more controlled trials. But I think it can be helpful to leverage in those patients who have, you know, comorbid obesity, sleep apnea, diabetes. And I find it helpful to kind of work with the patient's primary care provider to access those medications if they're a good candidate.

Dr. Peña-Robichaux:

One of the interesting things we were chatting about with one of our groups was, you know, when you do have patients with PCOS and are on these hormonal therapies and they decide they want to try to get pregnant, you know, often you get the question, 'Well, what's going to happen when I come off my medication? What's going to happen during the pregnancy?' And just, you know, it's talking about how to counsel about that. I mean generally, pregnancy, I always tell patients is we really don't know. Some patients actually go into remission. Some patients, things kind of stay par for the course. Some patients might flare. But just, you know, letting them know no matter what, you're going to support them. There are therapies that they can continue if they need to during their pregnancy.

And also just being aware that patients who have HS and who go through pregnancy, we do see kind of higher rates of some complications with these pregnancies, higher rates of gestational diabetes, preeclampsia, need for Cesarean section, things like that.

Dr. Horissian:

Piggybacking off of your comfort in the exam room technique, I think the chuck and the pillow are really good. One thing my mentor taught me was to do the exam, and if you're not going to inject or do a procedure on the patient, to step out, let them change into normal clothes, and then you talk like a normal human instead of a naked patient. So that's something I've incorporated to help it be a little bit more normal.

All right, I get to talk about the fun stuff. What are we going to do about all the HS that we've now diagnosed? Okay, so this first slide compares our three FDA-approved therapies. So we have adalimumab. It's usually dosed Q weekly. It's approved 12 and up, and then the dosing is right there on the slide. So you have 160 mg on the first day and then 80 mg Q15, and then you can do 40 mg Q weekly or 80 mg every 2 weeks. I personally do the weekly dosing. It is approved in our pediatric patients, and the dosing is on there too.

Secukinumab, a different mechanism. So it's an IL-17A inhibitor. It's dosed every 4 weeks usually, but you can go to every 2 weeks in patients that need that. And then one new thing this month is that secukinumab was approved for 12- to 17-year-olds, so we now have two agents approved for our adolescent patient population. I treat a lot of kids, so it's great to have more options.

And then bimekizumab is the newest on the block. It's an IL-17A and F inhibitor. It's dosed every 2 weeks and then it's every 4 weeks after the introductory 16-week period.

Looking at who's a good candidate for biologics. So I really base this off of the kind of the questions that we've been getting posed, which is what defines moderate or severe. And if somebody is having frequent flareups, the flareups are interacting with their life, they're not able to have sexual intercourse, they're not able to live a normal life and go to the gym or go to a restaurant because of fear for leaking or the flareup's every week and they never have a free period, you know, those are all reasons to do it. If somebody has draining tunnels, that's something that's going to need a stronger treatment. And the earlier we start these, hopefully the earlier we can kind of prevent the scarring and tunneling and progression of disease that comes with it.

You have to take into consideration a bunch of things. What's the patient's preference? What's their age? Do they have other comorbidities that might dictate your treatment options? And you kind of marry all those together like we would for any of our patients to find out what's the best option for our patients.

So delving into the data a little bit more, adalimumab was the first one to get approved. This is their primary endpoint here. And just to take a pause, the primary endpoint in most of our HS trials is the HiSCR50. So what that means is that there's a reduction in the number

of abscesses and nodules by 50% and no new draining tunnels. It's not a perfect measure, but it's the best we've got.

So here you can see that at week 12, that was their date of primary endpoint. In PIONEER 1 and PIONEER 2, the treatment arm was significant, so about 41% to 58% of patients achieved a HiSCR50 compared to placebo. Why is there a difference between PIONEER 1 and PIONEER 2? Well, one potential is PIONEER 2 actually allowed patients to be continued on oral antibiotics, and so that might be why PIONEER 2 is a little bit higher. It's also probably more realistic of what we would do in practice. You're not just going to pull somebody off because you're starting a biologic.

This is the safety data from that trial for their phase 2 part where they randomized people that were in placebo back to the treatment arms and placebo and people that were on treatment arms to placebo, and there weren't any big new safety signals from the original 12 weeks.

Moving on to SUNSHINE and SUNRISE, which were the secukinumab trials. This is 52-week data. Their primary endpoint was at 16 weeks, so a little bit different than the PIONEER trials. They also required five lesions instead of three, and they had two different dosing regimens compared. And you can see that about 60% or so of people on the treatment arms get to that HiSCR50.

Looking at the safety of the drug, again, no huge safety signals by comparison to placebo. Did have a few more fungal infections, particularly candidal infections, which makes sense with the mechanism of action—IL-17—important for fighting off fungus. Most were treatable. And IBD cases in the SUNRISE trial.

And then moving on lastly to bimekizumab. So bimekizumab, similar structure trial in that patients had to have at least five lesions in two different locations. Two different dosing regimens were done. And in this first dataset that I'm showing here, this was where they had a more stringent criteria, so patients could not be given any antibiotics or increased on their antibiotic dose or else that was considered a treatment failure. And you see about 50% of patients or so achieved a HiSCR50.

When you look at longer-term data and a little bit less stringent, you can see that that number goes to almost 80% for HiSCR50. And one of the coolest things to highlight here is that for the first time, we're talking about HiSCR75. So this reminds me of psoriasis, where we used to talk about PASI 75. Now we're talking about PASI 90s and PASI 100s. So I think the bar is getting raised in our clinical trials as to what we want. We don't just want a 50% reduction in abscess and nodules; we're looking at what happens when you have 75% or 90 or 100. So I'm really excited for what's to come.

Looking at safety of bimekizumab, again, very similar safety profile to placebo. We have more candidal infections in this one.

And really exciting to me is what's to come. So we have here listed all the later-phase medications, and you can see that there's a bunch of different mechanisms. We've got JAK inhibitors. We've got an IL-36 inhibitor. We have an oral BTK inhibitor. We have another IL-17A and F inhibitor. We have a topical agent. We have oral agents. We have subcutaneous agents. So it's an exciting time for HS. There's a lot that's coming out and hopefully a lot more options than we'll have other than the three that we currently have.

These are some oldies, newies, and goodies. I'll try to touch on some of the questions that you all had while I'm doing this slide. So one oldie but goodie can be infliximab. It's not FDA approved for HS. The dose that most people find helpful is usually higher than the dose we use in psoriasis, somewhere between 7.5 and 10 mg every 4 to 6 weeks. That can be really hard to get approved depending on the insurance, but I find infliximab very helpful for those patients that do respond to a TNF, but maybe bigger in size and more a weight-based dosing would be more helpful.

Roflumilast—it's not universally helpful, but there was a question in the chat about using apremilast. And I used to use apremilast until I found out about roflumilast. Roflumilast is generic, so it's available for pennies on the dollar, and people can use GoodRx or other things to get it. It doesn't work for everybody. I don't think it's as heavy hitter as everything we've talked about so far, but it can be a good intermediate or a good add-on to therapy.

And then we kind of touched on this with the Q&A session, but spironolactone, metformin, GLP-1 agonists are all helpful. And the biggest takeaway with HS is you don't have to use one agent. You can use multiple agents and tack them on and tackle different parts of the HS pathogenesis.

I'd be remiss—I do a lot of surgery. Does anybody do HS surgeries in this room? Okay, handful of people. Awesome. Hopefully after this talk, you'll be doing even more. But I think medicine is a really helpful tool and is one of the most important things, but surgery can also be really helpful and something that's in our wheelhouse to do small procedures like derooings or larger procedures like excisions. And I find it best for tunnels that are not responding to biologic therapy or abscesses and nodules that keep recurring despite our best therapies.

My tips if you're ever going to do an HS procedure is it's going to take more anesthesia than if you're taking off a mole on somebody. So

anesthetize well.

Don't forget about secondary intent. You don't have to close things up. The body is really good, particularly in intertriginous areas, at healing. And after the surgery, people have limited morbidity because they can move their arms. They're not tacked down with sutures.

Petrolatum is key. The more moist the wound can be, the better it'll heal and the less pain and irritation patients will have.

And I will rarely jump to surgery first. I think most people need a medication first so that we can quell the disease and then surgically remove what's gone. I also think this helps with surgical outcomes, and they have less chance of recurrence and quicker healing.

There was a good trial called the SHARPS trial, which is helpful if you ever send a patient to a surgeon. A lot of times they want to stop the biologic because they're worried about infection risk and those kind of things. You can always show them this study, which showed that the treatment arm that had adalimumab plus surgery compared to placebo plus surgery did better in all regards, both surgically, medically, and there were no increased risks of any infections, complications, or bleeding after the surgery. So I keep all my patients on their treatments when I do surgeries.

Okay, so shared decision-making is important, particularly when we're picking medications. I might think I have a good plan in my head, but if the patient doesn't want to do injections, we have to tweak my entire plan. So it's important to have that shared decision-making. Let patients lead it. I usually tell patients they're the captain of the ship and I'm their first crewmate. I give them the options, and then they tell me which direction we're going. You know, you have to think about economic needs. I think you have to learn their goals, their preferences, earn their trust. Don't do the largest surgery you've ever done for the first time. Start small, build their trust, and go from there. And then having help with medication access and support can be really helpful.

And that takes us to patient case 3. So this is a 17-year-old girl diagnosed with HS 4 years ago. Her symptoms were managed with episodic antibiotics, and she has on her buttocks, vulvar regions, and inguinal areas a lot of indurated plaques that have pustules on them. She has focal taut nodules, a lot of drainage when you press on it. She's getting ready to start college, and she's worried about managing her condition when she's going to be living in a dorm and adjusting to a new academic environment. She has frequent flares and persistent pain, which have already caused her to miss classes and social activities in high school, and she says she's excited about college, but she's scared her HS is going to hold her back. She doesn't want to miss out on opportunities and feel different from everyone else.

So for our small-group discussion, you know, what management decisions would you encounter with this patient's case? What factors are you considering when you want to select a treatment for this patient?

Dr. Peña-Robichaux:

Hello, virtual audience. This is Venessa here, and I'm looking through your questions. Let's see. Okay, I have a question here. It's two questions. Do you find certain drug classes work better for certain morphology types? For example, IL-17 greater than TNF for draining tunnels? And then second question, Hurley is a flawed system, especially for assessing response. Do you use a different scale, like the HS-PGA or IHS4 in clinic? Oh, those are great questions.

So with regard to certain morphology types, it's a great question. I think right now, we don't have data to support using specific biologics based on morphology types or clinical phenotypes. I will say though, as I was referencing in the first part of the talk when we talked about inflammatory versus follicular phenotypes, when I do have patients that have more of a follicular phenotype where you see more comedones and kind of acneiform lesions, I do tend to add on systemic retinoids as adjunctive therapy, and I find that sometimes to be helpful. But otherwise, I think it's difficult to say, you know, is this patient going to respond more to an IL-17 or TNF? And often what I tell patients is, you know, we just kind of have to give it a try and see how it goes. I do know that there is some evidence to suggest that potentially for young, thin males who have that phenotype where the buttocks and groin area is involved, there's some evidence to suggest that IL-23 inhibitors may be helpful in that scenario. But again, those are off-label use and difficult to actually get approved.

And then the second question with regard to using Hurley, so yeah, agreed, Hurley staging, you know, I don't find it too helpful other than just kind of describing the type and extensivity of the lesions. It is definitely not associated with severity. But in my own clinic, I do do HS-PGA scores. I like that versus IHS4 because it's very easy to calculate, and it's a great way to assess whether or not your patient's improving. And for those of you who have never taken a look at it, I would take a look at it. It's really easy to implement. It's just based on, you know, how many inflammatory nodules they have, how many draining tunnels they have. It's pretty straightforward.

The other thing I do, is I do also track pain scores. I think that can be really indicative. But again, you know, HS, it ebbs and flows. One day you're fine, one day you're not fine. And so it's difficult, right? Because we're just catching our patients at a moment in time. So I also, of course, ask questions to patients at follow-up regarding, you know, how many flares have you had since your last visit? Have you gone to the ED? Things like that can be helpful to assess, you know, improvement and clinical improvement.

Okay, let's see if we can find another question. Let's see. What is the impact of inflammation on other body systems? That is a good question. So, you know, the way I explain HS to my patients is that even though we think of HS just as a skin disorder, it is more than that, right? We do know that patients with HS have these comorbidities. We see higher rates of cardiovascular events, heart attack, stroke, pulmonary embolism. I have had a few patients of mine—actually more than a few—have these comorbidities. We see, you know, metabolic syndrome in these patients, these inflammatory, you know, seronegative spondyloarthropathies, things like this. So even though, yes, you know, we think of this as a skin condition, I kind of explain this is not just a skin condition. This is kind of a systemic inflammatory condition. And it's also important to explain that to patients because when you have a patient that you're having a discussion about potentially like a biologic, for example, I do explain that, you know, not only will this help your skin and, you know, we have data from example from psoriasis patients that going on these meds potentially could also help with systemic inflammation and therefore decrease, you know, the risk as it relates to these other comorbidities.

Let's see. Another question. Okay. So with regard to this case that we just discussed, consider her age and social situation and experiences at college, how far is college from HS treatment? Does she need debriding before or after starting biologic? Can she give her own biologic shots? Yeah, so these are really good points. You know, when you have someone that's going away to college, you have to think about all of these questions. You know, if they're going to be giving their own shots, for example, do they have access to a refrigerator? You know, if the patient is severe enough, you know, where you think they're going to need something more than an injectable, something like infliximab, is there an infusion center nearby? And then, you know, as far as surgery goes, I do have some patients that, you know, are surgical candidates. And timing is really important, right? So you don't want to do a surgery during spring break right in the middle of the semester. So we plan for these things. So having those discussions is really important.

Okay. Real quick, I see a question about, can you talk a little bit about sweating? I often think about Botox in my more mild patients, but maybe I should be thinking it in severe patients too. With regard to Botox, I ask patients if they feel like sweating exacerbates their symptoms. We also do glycopyrrolate as well sometimes, and some patients find this helpful, but I don't offer it to all my patients unless they think this is an exacerbating factor for them. And the other question says, maybe we'll get to this, but can you talk about goal dose of metformin for HS.

Dr. Horissian:

So recapping a little bit, I think there was a lot of really good discussions, you know, particularly to this patient. Again, it's an adolescent pediatric patient. How many options do we have available biologic-wise? Does anybody remember? Two.

Great. Okay. But where would she keep that biologic? In a college mini fridge? Who's her roommate? Is she going to take it? Is it safe? You know, what other things is she considering? Maybe she's considering starting birth control now that she's going to college. And so there's a lot of different factors to take into place. What if she's going out of state? When is she going to see you again? Or you're going to have to schedule visits around breaks? There's a whole lot of social factors to consider in addition to just picking the right medicine for her. And I think depending on the patient, she may have a different opinion on, you know, 'I want to start birth control and spironolactone' or 'I want to go really hard and start a biologic.' And I think that individualizing that to the patient's needs and wants is the best way to do it.

Do you guys have any additional points?

Dr. Barnes:

I think one of the things that came up in our group was like surgery, like, what if we just did it now before she left and maybe that would take care of a lot of what she would have to deal with. But we kind of talked through how when things are really inflamed, if there's not a medication on board, surgeries can be bigger, more likely to have recurrence. So it's really helpful. And we wouldn't do that for an inflamed cyst, right? We would typically try to inject with an ILK or give them some doxy beforehand so they get the better outcome. And that's kind of how I think about HS surgery as well.

Dr. Peña-Robichaux:

Going along those lines, I sometimes feel that our pediatric patients, because we're seeing them earlier in their disease, sometimes the parents are kind of pushing for surgery more because they see it as like a quick fix, and I always find it a difficult conversation.

Dr. Horissian:

I agree. I think the flip side to that is sometimes people are scared to treat kids, and I treat a lot of kids, so I love kids. And I think if you see somebody progressing quickly or you see somebody who has severe disease, don't be scared to start them on a biologic. They're approved now, so you should have the FDA behind your back to say that it's okay. But it goes for that point that the earlier we can start something and change the trajectory of their disease, the better.

Okay, so key takeaways of everybody's talks. We've got HS is a systemic inflammatory disease. It's got a lot of comorbidities, including

metabolic disease, inflammatory bowel disease, inflammatory arthritis. So you need to plug them in with multidisciplinary team. Diagnosing early before tissue destruction occurs is really, really helpful. The earlier we can be aggressive about management, potentially the better the patient's long-term outcome will be. I really think of biologics as a disease-modifying agent for HS, and I think it's helpful to think of it that way because HS profoundly impacts their quality of life. It's not something they're just going to have for a year. It's something they're going to have for decades. And there's pain, stigma, and mental health associated with that. Educating patients and having them in the decision-making process is critical to coming up with a good plan. And there's a lot of new and emerging therapies that are expanding our armamentarium, and I'm really excited for the future.

So now we're going to do the post-tests. So this is back to the iPads here. We've got a 28-year-old patient presenting with recurrent painful nodules in the axilla and groin that occasionally drain and recur over time. He's concerned he may have HS. Which of the following clinical presentations is not suggestive of HS?

All right, looks like most of you got it here. So definitely in the post, we're doing really, really well with 95% of people getting the correct answer here. So the first episode of an acutely expanding abscess on the lower extremity associated with fevers and chills. So HS classically is a recurring disease. You know, by quick definition, we say you have a typical lesion in a typical area happening at least twice in a period of 6 months. And so having a first episode of a lesion that doesn't fit the description outside of the typical area doesn't fit, whereas A through C would all fit.

Okay, post-test 2. We've got a 44-year-old man with Hurley stage 2 who's got recurring painful nodules and draining lesions in the axilla despite previous therapy. His dermatologist explains that dysregulated inflammatory pathways contribute to disease progression and are targets of emerging therapies. Which of the following best explains how IL-17 plays a role in the pathogenesis of HS?

Awesome, so this one was a little bit of a stumper here, but we definitely got some improvement from pre- to post-test. So just highlighting, you know, HS is a follicular-occlusion disease that has rupture triggering IL-17-driven inflammation, and that promotes the downstream of IL-17, 23, TNF alpha, and IL-1 beta.

Moving on to post-test number 3, we've got a 38-year-old Black woman with history of hypertension, PCOS, diabetes, lupus, and Hurley stage 2 HS who presents with nodules, abscesses, and tunnels that continue to drain despite topical and systemic antibiotics. She reports significant quality-of-life impairment, including pain and postinflammatory hyperpigmentation. Her dermatologist is considering escalation in therapy. Which of the following would be the next step in managing her disease?

Okay, little bit of a trick question here, but the correct answer is IL-17. You're technically not wrong by picking a TNF alpha. You could do that, but this patient has a history of lupus, and so generally we try to avoid TNF alpha inhibitors in patients that have lupus. And so since we do have another option of an IL-17 inhibitor on the screen here, that would be a superior option to start the patient on.

Okay, post-test 4. We've got a 36-year-old man visiting the doctor's office for a follow-up. His HS progresses to Hurley stage 3 disease. He's failed treatment with adalimumab, isotretinoin, and infliximab. He's interested in trying one of the recently approved IL-17 inhibitors. While counseling your patient about treatment expectations with an IL-17 inhibitor, which of the following safety considerations would you discuss?

All right, great. Almost everybody got this one right. So risk of inflammatory bowel disease. So the IL-17 inhibitors were tried to use for inflammatory bowel disease years ago, found that it made the disease worse, so they stopped those trials. And in most of the IL-17 trials nowadays, we actually screen for IBD beforehand and don't include patients with IBD beforehand, and then we look for if a new development of IBD happens during the trial. But generally you would avoid IL-17 inhibitors in patients with inflammatory bowel disease.

Okay. And last poll here. Do you plan to make any changes to your clinical practice based on what you learned in today's program?

I lied. That wasn't the last one. Okay, please take a moment to enter one key change you plan to make in your clinical practice based on this education.

Well, that's okay. We can move on to the Q&A session. I saw one question that I meant to answer while I was talking about medicines, but one question was about TNF inhibitors. What's the highest dose of adalimumab that you can do? There's probably a safety answer to that. I don't know what that is, but I have had patients that have needed double the approved FDA dose. It's sometimes hard to get approved now that we have other options that you can switch to, but that's what I've used before. I haven't gotten above that, so that's usually my max dose.

You can check an adalimumab level and antibody level, which can be helpful in determining if that's the issue. Sometimes people may metabolize it quicker or be bigger and need more medicine. And so you can look for a drug level and make sure that they're not just eating away at their drug with antibodies and escalate the dose if their drug level is very low. The question as to what the correct drug

level should be is also kind of questionable, and we think it should be higher than that in inflammatory bowel disease when looking at HS patients. So generally above 20.

Dr. Barnes:

The thing that I usually will check for as well is if they notice really good response in the loading period that went down when they went down to like 40 mg weekly, then that gives me a hint that like, oh, they just needed some higher doses. But if they didn't notice much during loading, they're not really doing much on 40, and you're going to have to fight tooth and nail with insurance to get the 80 mg weekly, it's probably worth it to just try something else. And especially if you're worried about like are they getting enough of the medication, then infliximab is a great option. And a lot of patients like the idea of, 'Wow, this medication is actually- I'm getting the amount for my body,' which is what I explain to them; we're giving you medicine that's based on your body size so that it's the right dosage, and tends to go over pretty well.

Dr. Lee:

Yeah. I use 80 Q week often as well, and the HS Foundation has a wonderful prior-auth letter that I found super helpful, so you can access that and just fill in your patient's information and send that often. And that's been really helpful in terms of getting coverage.

Maybe question for in terms of post-deroofing infections and hypergranulation tissue, how do you counsel on wound care to optimize healing?

Dr. Horissian:

So I think everybody that does a deroofing will have a different answer to this slightly. I have seen zero infections in the hundreds and thousands that I've done and our residents do. So I think the infection risk is very, very low. I think a granulating HS wound just is less likely to get infected than if you cut out a mole on their back.

But my usual postop is Vaseline, Vaseline, Vaseline with a good bandage to hold that Vaseline in place, and then I'll add timolol eye drops and clobetasol ointment if I see a lot of hypergranulation tissue to kind of help prevent that.

Dr. Barnes:

Yeah.

Dr. Horissian:

Timolol. They're eye drops. Timolol—I forget what the second word to it is, but timolol eye drops—Timoptic was the brand that we usually use. And you do 1 or 2 drops depending on the size of the wound in the wound, and it helps with hypergranulation tissue.

Dr. Peña-Robichaux:

Yeah, I typically inject intralesional Kenalog into the deroofing site right as soon as I'm done with the procedure and also have the patient apply clobetasol into the wound with their dressing changes for the first week. And I don't know if I've ever gotten hypergranulation. And same answer, I've never had an infection with deroofings. I wonder if what's going on is if it's just inflammatory drainage maybe, recurrence potentially.

The other thing too is that I find when patients are on biologics, they actually heal way quicker, less postop pain. It's just so much less complicated when you have these patients who have very focal tunnels or lesions and they just want you to just, you know, deroof it. Oftentimes those will, you know, have more postop complications, take longer to heal, and so I try to encourage some sort of anti-inflammatory treatment if they're going to have a surgical procedure.

Dr. Lee:

There's a question on zinc and if we kind of routinely recommend zinc. I think zinc can be helpful. I find it more helpful as an adjunctive therapy or maybe something you can implement for kind of milder HS. Typically for zinc, I'll recommend 90 to 100 mg of elemental zinc. And what's important is when you give zinc, you also have to let your patient know that they should take a little bit of copper, usually at a ratio of like 10:1. But it is something easy they can find over the counter, generally pretty well tolerated. Sometimes can cause a little bit of GI upset. So if it does cause GI upset, you can also recommend it in like a gummy form, and that can be helpful.

How about laser hair removal? Any thoughts on laser hair removal for HS?

Dr. Horissian:

I use laser hair removal a lot. I don't do it as much for like stage 3 disease, although there are some studies showing it can be helpful. But definitely stage 1, 1-1/2, 2, I think it's really, really helpful. I think in the US, coverage is a big issue for this, and there's a lot of efforts to kind of make it an official treatment for that and all that. But I think it can be helpful.

Dr. Peña-Robichaux:

Yeah, I love using laser removal in addition to kind of earlier milder HS in the postoperative setting. So when you have patients who have like Hurley 2 or 3, very widespread involvement of the axilla or groin, they have surgery, they're on a biologic, I often will recommend adjunctive laser hair removal preventatively just kind of in addition. Because especially if they're going to see someone in plastics where they have these flaps, you know, so you're still having skin on skin, you're still having follicles around the area, you can go and kind of laser around those areas, and I think it works well.

Dr. Barnes:

And I think to the point about cost, because a lot of times we are advocating for it to be covered by insurance, but when that's not possible, like I've partnered with some like local laser hair-removal like groups. So like SEV Laser, for example. Like I personally went there myself, explained to them what HS looks like, what to expect. They had like the MD of the GentleMax that we use at Stanford as well. So, you know, safe for darker skin tones because it had the 1064, but then also had alexandrite. So there are things that if you're noticing a lot of your patients aren't getting things covered, then you know, a Groupon of like 6 sessions for Brazilian and axilla for only \$360 in the Bay Area is pretty hard to beat.

Dr. Peña-Robichaux:

So there's a question about comparing secukinumab and bimekizumab. So, you know, secukinumab blocks IL-17A and bimekizumab blocks IL-17A and F. And if you look at kind of the data, it does seem that bimekizumab seems to have better efficacy. I think in the real world, at least from what I've seen, I think that is true. The way I kind of describe it to my patients is that it's kind of like a stronger version of secukinumab. However, I have seen definitely more side effects associated with it. Specifically, I am seeing a lot of patients with candidal infections, thrush, and I've had actually two patients with esophageal involvement who had to go to the emergency room. It was really, really bad. And then these kind of psoriasiform rashes.

And what's interesting is that these side effects usually occur pretty quickly, like usually within the first 1 to 2 months. But so at least from what I've seen, if you go beyond the loading period of a bimekizumab and you're responding and you haven't had one of these side effects, I think generally you're kind of in the clear. But that's just been my experience.

I don't know about you guys?

Dr. Horissian:

I think it's really hard to compare the trials because they're designed differently. They're in different time periods, different patient populations. And then what's your comparison? Are you comparing how many reached HiSCR50 because they might have different placebo rates? And so then maybe you're looking at the delta to placebo. So I think it's really hard. I think you can look at it one way and come up with one medicine being the best and look at another way and I think it's some of it's number playing. So I think it really depends on the patient. Like some people respond better to TNFs in my opinion, and some people respond better to IL-17s. I can't figure that out until I give it to them. I haven't found the answer to that. But-

Dr. Peña-Robichaux:

Yeah, I also think that if you have a patient on secukinumab and you have them on Q2-week dosing, which is kind of like the, you know, highest dose you can do, and you've had a partial response, at least maybe 50% improvement, but they're still having breakthrough flares, those are the patients that I feel better about trying something like the bimekizumab because, you know, they're already responding. Just kind of how you were talking about earlier, if they've had a partial response to adalimumab, then maybe you check levels, but you would consider increasing the dose. So I take that into account as well.

Dr. Barnes:

And I think another important question that we got much earlier is the role of imaging in assessment, especially in the pelvic area. And I think like that goes to extend to like perianal area. I'm sure all of us here have gotten a lot of patients who, you know, manage HS, and then you evaluate them and it actually doesn't seem like it's HS. It looks like it could probably be Crohn's or like maybe someone has like a perianal fistula.

And so one of the key things that at least I think about is the discharge from HS is usually not as copious as like if someone has a fistula. So if it's like a higher volume and like a little bit more malodorous, that's definitely a case to just get an MRI since a lot of times like these patients might be referred to me for surgery, and the last thing I want to do is get into a fistula.

The other thing is, is if it's particularly close to the perianal verge, I at least will try to just get some imaging because of that higher risk.

And then also just getting other history like we were saying with the comorbidity screening. There are some cases where there's cutaneous Crohn's that I think it's— if I remember from like Maloney or something like 30%—like sometimes people can have cutaneous Crohn's before they even have GI symptoms. But for a lot of patients, they maybe have both. So making sure that you

screen for that.

And then occasionally like biopsies can be helpful. If I feel like this looks a little bit more like knife-like fissures from cutaneous Crohn's, the granulomas as we expect from the pathophysiology of HS should be around hair follicles. If you have noncaseating granulomas that are not associated with the hair follicles, for me that helps me think a lot more about Crohn's and then make sure that we get GI to do the endoscopy, the colonoscopy, and complete workup.

Dr. Peña-Robichaux:

Yeah, there are these patients who, you know, will just have perianal involvement. And if they have no involvement anywhere else, you always want to think about possibly Crohn's. I've had these patients where you go and they see GI and they do the colonoscopy, they don't have Crohn's, they don't have lesions, and they'll just have these perianal fistulas. And I feel like these patients are kind of their own clinical entity, you know. I feel like they're harder to treat. But the other thing I was going to add is besides sending to GI, I have done like fecal calprotectin too. I've sent for that because I find that it's not a perfect test, but sometimes if that's really elevated, that could indicate something as well.

Announcer:

You've been listening to CME on ReachMD. This activity is provided by Clinical Care Options LLC, doing business as Decera Clinical Education, in partnership with the HS Foundation. To receive your free CME credit or to download this activity, go to ReachMD.com. Thank you for listening.