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What Are the Latest Updates in Lupus, Fibromyalgia, & Sarcoid?

Announcer:
You're listening to Conference Coverage on ReachMD, captured on location at the Congress of Clinical Rheumatology's Annual Meeting in Destin, Florida. Your host is Dr. Madelaine Feldman, Clinical Associate Professor of Medicine at Tulane University Medical School and Vice President of the Coalition of State Rheumatology Organizations.

Dr. Feldman:
This is Dr. Madelaine Feldman talking to you from Congress of Clinical Rheumatology. I'm here with Dr. Stephen Lindsey, and he's going to give us a few of the highlights from Dr. Michelle Petri's nonrenal lupus talk.

Dr. Lindsey:
Dr. Petri's talk was excellent. I think that I learned that following C3 and lupus anticoagulant are very important parameters when you're looking at cardiovascular mortality—I'm not sure that I really followed that carefully in the past—and trying to normalize those and keep an eye on those will help decrease your risk of a patient's biggest comorbidity in lupus. A second point she made over and over again was that we use too much prednisone in lupus, and I think I need to change my practice—when

people flare, trying to keep the dose to 20 to 30 mg for the flare and not higher, 60 mg, and then to taper quickly. The dose of prednisone is directly related to the cardiovascular mortality in lupus.

Dr. Feldman:
Dr. Petri also gave a lecture on Update in Lupus Nephritis. What are some of the high points of that lecture?

Dr. Lindsey:
I think the high points of that lecture were for us to realize that every nephron counts, that from the very first flare, people begin to lose their renal parenchyma, that we need to be very quick in treating these patients, that we need to focus on their blood pressure and try to keep their blood pressure in the 120s or lower and not higher. We need to focus on their proteinuria and use ACE inhibitors much quicker and be following proteinuria every visit, and if we do that, we can save a lot of people's kidney function.

Dr. Feldman:
There was a talk on fibromyalgia by Jarred Younger. What were the high points on that one from the Congress of Clinical Rheumatology?

Dr. Lindsey:
I know that most of us rheumatologists hate or shiver at the word fibromyalgia, but Dr. Younger did a great job of presenting some new data on microglial cells in the central nervous system. And these cells appear to be the primary factor in hypersensitization syndrome in fibro, and he showed how they will go from a resting state to an excited state, and how when you think about therapies, trying to place the microglial cell back into its resting state, and that tends to give us a much calmer CNS and not so hyperexcitable. He gave a lot of data on naltrexone, low-dose being a very attractive alternative for that, and I think it's something we should all think about using. And a lot of experimental data on trying to push microglial cells back to a more normal or what he calls protective state may be the future of fibromyalgia.

Dr. Feldman:
There was also a very up-to-date talk on IgG4 by Arezou Khosroshahi, and what were some of your high points that you found in that lecture?

Dr. Lindsey:
I think we all have been inundated with IgG4 disease as something we need to know about. It seems to mimic a lot of our diseases, and I think what I found interesting in this talk was that our expectation that having a high IgG4 level as a marker may not be true. Our expectations of having increased plasma cells being a very diagnostic marker of IgG4 cells in the tissue may not be true. It turns out that the

best criteria is the pathology showing certain characteristics like obliterative phlebitis and the storiform fibrosis, and so you really can make a diagnosis of IgG4 with normal IgG4 levels and with no increased IgG4 plasma cells in the tissue, so I think that's pretty interesting.

Dr. Feldman:
Yes, that's very interesting. Were there any other points in the IgG4 lecture?

Dr. Lindsey:
Yes, he made 2 other points, and that is that these patients almost never present with fever and that they always respond to steroids.

Dr. Feldman:
Thank you. And finally, there was a very good talk on sarcoid: What's New in Sarcoid? by Dr. Daniel Culver. What were some of the high points in that one?

Dr. Lindsey:
I think Dr. Culver did a great job of covering sarcoid from beginning to end. I think what I found most interesting was that we are probably using too much steroids for too long in sarcoid, that actually the worst case is if you just kind of manage them carefully and with low-dose prednisone, they will resolve on their own. His point was that the worse it looked the better chance it had of going away, and that with sarco we better have an adage of "do no harm." So use lower doses, do shorter courses, and just kind of follow for anything, the serious organ disease, but just because they have it, it's a lot of times best just to follow it and tell the patient you're going to be on top of them but not to overtreat them.

Dr. Feldman:
Did he discuss using methotrexate or TNF inhibitors in these cases, and if so, what were the types of cases that would benefit the most?

Dr. Lindsey:
Yes, he did. So, if you had more serious skin involvement or multiorgan involvement, he did use DMARDs. The most common he used would be methotrexate. And he did find that infliximab and Humira both had good data but that Enbrel did not work for sarcoid.

Dr. Feldman:
Great point. Thank you so much, Dr. Lindsey, for giving us those nice synopses of those lectures.

Announcer:
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