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Autoantibodies in CIDP: Prevalence and Diagnostic Implications

Announcer:
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Dr. Silvestri:
This is CME on ReachMD. I'm Dr. Nick Silvestri. Joining me today is Dr. Jeff Allen.

Jeff, let's focus on autoantibodies in CIDP. What can you tell us about their prevalence and diagnostic implications?

Dr. Allen:
Well, that's a great question and one without such a clear answer. I guess the place to start is to think about what the evidence for autoantibodies in CIDP is. And it comes from a couple of different places. One are some older experiments and animal experiments, where healthy animals were given serum from animals with disease, and those experiments were able to reproduce some of the symptoms of CIDP in those previously healthy animals and also some of the electrophysiologic changes. So those passive animal studies suggested that there's maybe some antibody that's playing a role in the pathogenesis of the disease.

Another line of evidence comes from some immunohistology studies, where if you look at serum with IgG and also IgM and look at staining on nerves, you can see some staining usually around the nodal and the perinodal regions of IgG staining in patients when the serum is applied to those nerves, suggesting that there's some antibody, although the specific one is often not known.

And then the third line of evidence is just what patients tend to respond to with CIDP, which is IVIG and plasma exchange. So plasma exchange probably takes on a lot of things other than just antibodies, but thinking that pathogenic antibodies are probably removed and, clearly, some patients do better with plasma exchange suggests there is an antibody that's playing a pathogenic role.

Having said all of that, in the vast majority of patients, we don't have any identifiable antibody. Over the years, there's been some antibodies that have been observed with different myelin proteins like PMP, but largely, those antibodies have not been reproducible and can't be relied upon in large populations of patients that are, say, you do or you don't have the disease.

I guess the one exception to that is the discovery within the last 10 years or so, the neuropathies, the neurofascin 155, neurofascin 140, 186, and contactin-1, and also Caspr1. So those patients are actually split off now from CIDP and its variants, in particular because patients with those antibodies have some unique features. They tend to have a lot of tremor and ataxia, and in the case of contactin-1, they can get very sick very quickly with a lot of early axon loss.

The immunobiology of those antibodies is probably different than CIDP without an antibody, and the treatment paradigm is also somewhat different, where they tend not to respond very well to things like steroids or IVIG. They respond better to B cell-depleting therapy.

Dr. Silvestri:

Yeah, it's almost like we've gone about this a little bit the opposite way with myasthenia, right? So in myasthenia, the antibodies have been discovered and we've then started using therapies that really go after the antibody. In a way, in CIDP, it's the opposite way. We've used therapies that we know work on antibodies, but we don't necessarily know the antibody in most cases. And as you point out, with typical CIDP, we don't know the antibody, right? The antibodies you mentioned are nodopathies where they're kind of broken off now.

And I think you bring up a really important point. I mean, this is not just neurological trivia. It's important to make these diagnoses in the case of nodopathies because, exactly as you point out, there are implications for treatment.

How far away do you think we are from finding the antibodies in traditional typical CIDP?

Dr. Allen:
I wish I had a crystal ball with that. I know there's a lot of groups that are really interested in finding antibodies, probably some target proteins in the nodal-perinodal space that are of interest. But finding a specific one, a biomarker that covers the majority of patients with CIDP, is really a major unmet need. You mentioned myasthenia gravis. Obviously, it would be great to have a situation in CIDP like that, where 80% of people with MG have a known pathogenic antibody. We're probably a long ways off from that in CIDP, but hopefully we'll get there.

Dr. Silvestri:
Yeah, hopefully we'll see it in our career.

Dr. Allen:
Yeah.

Dr. Silvestri:
Well, this has been a great micro discussion. Our time is up, and thanks for listening.

Announcer:
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