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High-Efficacy DMTs: Transforming MS Care

Announcer:
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Dr. Obeidat:
This is CME on ReachMD, and I am Dr. Ahmed Obeidat. Joining me today is Dr. Mark Freedman.

Mark, what do you consider when selecting high-efficacy DMTs for multiple sclerosis?

Dr. Freedman:
Well, I think, first of all, we have to define what high-efficacy therapies really are, and I think all the more recently developed medications are higher efficacy partly because they've been shown in clinical studies to be better than some of the so-called platform drugs that we have, like teriflunomide or interferon, and so they're proven to be of higher efficacy.

The whole reason we're having this discussion is because, although they're higher efficacy in terms of controlling the disease, they do bring more risk to the table, and therefore there needs to be a benefit-risks kind of evaluation for each patient to say this is somebody that warrants a higher-efficacy approach than someone who may not. And there are factors, for instance, that might predict who those individuals are.

Dr. Obeidat:
So in your clinical practice, Dr. Freedman, when you think about patients, when they come to you in clinic, do you just treat everyone with high-efficacy DMTs, or do you have selection factors that are specific for the patient?

Dr. Freedman:
Great question, because just as I was saying, not everybody warrants the risk for these higher-efficacy therapies. We've known about a number of clinical and radiological and now biomarker profiles that tend to indicate someone with a more aggressive course, so they're the ones that we probably approach. If you don't want to apply that knowledge, then sure, just take a blanket approach and treat everybody with a high-efficacy therapy. But then someone's going to get it that probably doesn't warrant it, and with your luck, that'll be somebody who develops one of the problems that are associated with the drug. So the clinical factors, or the type of relapses, the number, the severity, the burden of disease on MRI that they come to the table with, and now biomarker neurofilament light chain that helps to indicate a poorer prognosis.

Dr. Obeidat:
And patients typically, if they are on high-efficacy therapy, their relapses are under control; their MRI inflammatory activity is, too, under control. Disease progression sometimes it's not as powerfully impacted. So what do you think about that?

Dr. Freedman:
No question. That is the ultimate goal, is to prevent progression, and now that we can define this a little differently with the words PIRA,

progression independent of relapse activity, we are seeing drugs today and trials that control the relapses and inflammatory activity 90% to 95%, yet it's not stopping this PIRA. And that'll be the next generation of, I guess, higher-efficacy therapies that could address that component.

Dr. Obeidat:
Most of the data that we get on this topic, high-efficacy versus other moderate or traditional treatments, comes from retrospective studies. But we know there are two, now, studies in the United States of America. One is called TREAT-MS, and the other is called DELIVER-MS. And these are trying to provide us with prospective data to look at this question specifically.

In TREAT-MS, we randomized patients into traditional treatments or into high-efficacy treatments, and then we follow them prospectively over time to see if there is a difference between the groups. And there is an equipoise here, there's a flip of my coin, I tell my patients, that you could be in this group or this group or we get to choose what medicine.

DELIVER-MS is looking at also brain volumetrics and other things as a primary outcome but with the same approach, randomizing patients to one of the groups.

So what do you think about these two studies that are going currently?

Dr. Freedman:
Well, they're very important for, obviously, in the United States, the insurers are the ones that are dictating who gets what drug, and they need the data that will come from these two studies to actually justify the use of high-efficacy therapies in certain groups. That's important, because with real-world evidence, you can only balance on propensity scores so much, and all of that is based on things we know. But gosh, there's so much we don't know. How to pick the right drug for the right patients, this is going to come out of these prospective studies, hopefully. We'll get a lot of good data and then we'll be able to refine our ability to pinpoint the right drug for the right patient.

Dr. Obeidat:
Perfect. Thank you so much. This has been a great, bite-sized discussion. That's our time. Thank you for listening.

Dr. Freedman:
Thanks, Ahmed.

Announcer:
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