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Released: 09/08/2023
Valid until: 09/08/2024
Time needed to complete: 1h 04m

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Evidence-Based Discussion of the New and Emerging DMT Treatment Strategies for MCI

Announcer:
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Dr. Sabbagh:
Hello, I'm Marwan Sabbagh, Professor and Neurologist Behavioral Neurologist at the Barrow Neurological Institute in Phoenix. Joining me today is Dr. Sharon Cohen and Dr. Richard Isaacson. Dr. Cohen, please introduce yourself.

Dr. Cohen:
Hi there. Hi, Dr. Sabbagh. I'm a Behavioral Neurologist. I'm here in Toronto, Canada. I'm the Medical Director of Toronto Memory Program.

Dr. Isaacson:
And I'm Richard Isaacson. I'm a Preventive Neurologist down at the Institute for Neurodegenerative Diseases in Boca Raton, Florida.

Dr. Sabbagh:
Thank you both for joining me today. Our question of this moment as we see this rapid change in our practice is the advent of the new disease-modifying, or DMTs, and monoclonal antibodies. Dr. Cohen, can you discuss the new therapies needed to treat mild cognitive impairment caused by Alzheimer's disease?

Dr. Cohen:
Sure, well, first of all, let me say there's a huge need to slow down this disease and particularly to do it at an early stage where people are still functioning well. So mild cognitive impairment, you know, there's some memory difficulty, people are frustrated, yes, but quality of life is generally good and people are still working banking, shopping, independent, whether it's traveling, whatever it is they do. If you can keep people at that mild cognitive impairment stage and prevent them from progressing to the dementia stage, that's huge. And what disease-modifying therapies are aiming to do is to keep people at these early stages longer.

So we have a drug with full FDA approval now, lecanemab, which slows progression of disease, both slowing decline in cognition, decline in function, and less risk of progressing from the MCI stage to the dementia stage of disease. That is really important. That's not a cure for the disease, but that is a substantial improvement over having no therapies for MCI, which was the landscape. And until very recently, we've had a symptom treatments for the dementia stage of Alzheimer's, but no disease-modifying treatments for Alzheimer's and certainly not for at the MCI stage.

So this is big progress, and we're expecting more drugs to have full FDA approval, and hopefully, in other geographies, regulatory geographies, like Canada and Europe, we will also have these drugs approved. So the landscape is definitely changing.

Dr. Sabbagh:
Thank you. Dr. Isaacson?

Dr. Isaacson:
Yes, I think you know, this is among the most common questions we're getting from patients, from my colleagues, whether it's in primary care or in neurology or psychiatry, geriatric psychiatry, geriatrics, you know, as a subspecialty and as a field, we need to become more comfortable with using these drugs, we need to - obviously we've learned the data, we've had, you know, a lot of us have had patients in the clinical trials. You know, now I've had several patients on the drug, you know, in the real world, but it's all about the learning. And I think, you know, in terms of the earlier you treat, the better the patient will do, especially predementia and mild cognitive impairment, that's really our sweet spot. Having patients understand kind of the complexities of, you know, that this isn't a pill, this is not something you take, and then you go home and that kind of thing. So I think when

I think about these new therapies, of course, we talk about efficacy, we talk about safety, but I think there's a lot of real-world connotations too. It's been interesting, different patients, you know, think about different things, have to time their lives differently, because you have to, for example, with the new drug, you're going to – lecanemab, you have to be in town, you can't maybe travel as much, you have to plan things. So I think this is a - it's not just a drug, where we talk about the pathophysiologic mechanisms, it's about how to plan one's life and how to structure the care and also structure the surveillance of monitoring for side effects.

Dr. Sabbagh:
This is super important that you both bring up to make this declare it - to emphasize what Dr. Isaacson and Cohen said, both lecanemab and donanemab are intravenous fusible drugs, donanemab is once a month, lecanemab twice a month. And the idea is that these drugs don't make you better, they make you less worse. If you look at the data in aggregate, both products slowed the rate of decline on a global instrument called the CDR-Sum of Boxes by about 28-29%, and donanemab reported recently in a subgroup that they had even better effects, more than 35%. But we know that it's not small, and it is clearly better than what was the status quo.

This is going to be important because in order to get a patient in the infusion chair, you have to have determined that the patient has Alzheimer pathology, meaning amyloid, either by PET or CSF, and hopefully very near in the future, by plasma biomarkers. But the commitment here is that you'll need surveillance and vigilance, and we'll talk about that in our next segment.

So, thank you both. And thank you audience for your participation.

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