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/what-is-immunotherapy/14654/

Released: 12/30/2022
Valid until: 12/30/2023
Time needed to complete: 1h 02m

ReachMD
www.reachmd.com
info@reachmd.com
(866) 423-7849

What Is Immunotherapy?

Announcer:
Welcome to CME on ReachMD. This episode is part of our MinuteCME curriculum.
Prior to beginning the activity, please be sure to review the faculty and commercial support disclosure statements as well as the learning objectives.

Dr. Agronin:
This is CME on ReachMD, and I'm Dr. Marc Agronin. I'm here today with my colleague Dr. Richard Isaacson.

Dr. Isaacson, there's been a lot of talk about disease-modifying treatments for Alzheimer's disease, more specifically, what we call immunotherapy. Can you tell us in general, what is immunotherapy? How did it start? Where are we going? And let's have a good discussion about what's going on now with these particular treatments.

Dr. Isaacson:
Sure. So immunotherapy targets specific pathologic proteins that build up in the brains of people with Alzheimer's. And the anti-amyloid antibodies have been developed over decades now, believe it or not, decades. There's a variety of them from solanezumab to gantenerumab to aducanumab, donanemab, lecanemab, there's just many, many. And basically, these drugs are given usually through the IV and given over a long period of time and target amyloid and try to reduce amyloid buildup in the brain. And then you can actually track amyloid and track cognitive function to try to understand whether these drugs may or may not be effective.

Now, in terms of the history of these anti-amyloid drugs and immunotherapy, in the past, decades ago, when people got the drugs, we actually didn't have a biomarker to test for amyloid, so people with a clinical diagnosis of Alzheimer's were getting these anti-amyloid drugs. And some people may have had an effect and other people didn't. But when we ended up looking at the data later, when the Alzheimer's biomarkers came to fruition, many of these people, you know, 20%, 30%, 40% – up to 40% in some studies – people didn't even have amyloid, so the targeting of the anti-amyloid drug really wasn't the right thing to be done in that patient.

So basically, where the field is gone is to do an assessment, does the person truly have amyloid, and then we can deploy an immunotherapy, either in monthly or bimonthly infusions, or also sub-Q injections that were also studied but didn't come to fruition just yet. And then basically, people can be followed through their cognitive function, their PET scans, and then, you know, once their amyloid is low or gone, maybe then we would stop these drugs. And it's, you know, these are really important questions.

So that's a general overview. But, it's really been quite a field and a lot of developments, including 1 that was FDA-approved within the last year and then 2 that have been submitted to the FDA.

Dr. Agronin:
Sure, and now we've also been researching anti-tau immunotherapy as well. The question over time will be, do we do one or the other? Do we do both? And that's where research is heading.

The controversy comes in as is that we aren't seeing people even when there's a significant reduction of amyloid, as an example. We're not seeing people getting better. But we are seeing, in general, a slowing of the disease process. And so really leads to this question of

what's a meaningful outcome for this? And we're seeing with data that there's variability in terms of who's getting it, what is their genetic background? What dosing are they getting? So for instance, with aducanumab, which, garnered an FDA approval, we know that with especially certain individuals, who are getting the higher dose, had a significant slowing and their disease course. And so the question is, how do we track that over time? When we roll these out in terms of actual market products, how does that play out? We need to educate people in terms of the role of these. So exciting developments. And, Richard, where do you think we're heading with these treatments?

Dr. Isaacson:
Yeah, I think we're headed to kind of a new frontier where these drugs, you know, some people may think they're, you know, terrific, and they're great. And other people say, “Oh, they only slow decline by 27% over a period of time; that's not really worth it.” Well, you know, I think patients and their physicians need to really have these honest discussions. And you know, these drugs can be expensive, they're time consuming. There are also safety issues, which need to be kind of monitored. But, you know, I think whether one believes that X period of slowing of decline versus Y, I think there’s going to be a lot of learning that needs to be done. I also think we're entering an area of personalized medicine where different people with different genes are going to have higher or lower likelihood of side effects. So I think personalized medicine in Alzheimer's is now arriving. And I'm excited for the years to come with these therapies.

Dr. Agronin:
Sure, I agree with that. I guess one key takeaway that we tell individuals is that, especially if there's early symptoms of what may be Alzheimer's disease, to get a comprehensive diagnosis and then explore whether immunotherapy is an option, either through a clinical trial or through an agent that's on the market. But if there's an opportunity, especially in an early stage, to slow this disease down, that can make all the difference over time.

So this has been a brief but a great discussion. Unfortunately, our time is up. To everyone, thank you for listening.

Announcer:
You have been listening to CME on ReachMD. This activity is provided by Prova Education and is part of our MinuteCME curriculum. To receive your free CME credit, or to download this activity, go to ReachMD.com/Prova. Thank you for listening.