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The Role of Biomarkers in Alzheimer's Disease

Announcer:
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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, biomarkers are emerging as really hot topics in Alzheimer's disease right now. And I wanted to ask you, you know, what are these biomarkers? What role do they play in Alzheimer's disease? And let's talk a little bit about what patients need to pursue in order to get at the best diagnosis.

Dr. Isaacson:
Sure. So I think most people may or may not be aware that Alzheimer's disease is actually characterized by a very specific set of neuropathological changes. We've known for a long time that the brain shrinks as we get older, and especially in certain areas related to Alzheimer's pathology. But much more specific than just shrinkage of the brain, but we also have biomarkers, AD-specific biomarkers that include amyloid, tau, as well as neurodegeneration.

And really where our field is going and where it really almost is today is the ability to assess these different types of amyloid ratios, different isoforms or different types of tau, as well as different markers of neurodegeneration. For example, neurofilament light, GFAP, there's a variety of new emerging biomarkers that we can now assess in the blood, as well as old, more traditional biomarkers that we're also able to assess through amyloid scans, which are positron emission tomography tests, or PET scans, as well as in the spinal tap.

So I guess what I would say overall is that there are a variety of different Alzheimer's-specific biomarkers that can now help characterize the disease better. It can also not just characterize is the person having Alzheimer's or a different neurodegenerative dementia, but also understanding a little bit more about where the person is on that long-term trajectory. For example, if there's some amyloid but there's not too much tau, or if there's amyloid and tau but not yet neurodegeneration, we can use these measures to understand really a little bit more about staging and what specific targeted therapies may be more appropriate.

Dr. Agronin:
At this point, I think people have questions about the differences between amyloid and tau and how they both fit in and are they equally important? Tell me your thoughts about that.

Dr. Isaacson:
The traditional model of Alzheimer's is basically you first have the amyloid and then you have the tau, and then you have neuroinflammation, and then neurodegeneration. There's also glucose hypometabolism. So there's a variety of pathologies that one can detect in the brain on a variety of different neuroimaging studies, spinal fluid tests, as well as blood tests or plasma tests. I think we can argue all day whether amyloid causes Alzheimer's or tau causes Alzheimer's or neither causes Alzheimer's and they're just markers of the disease and the definition of what a person has in terms of their neurodegenerative dementia.

I really believe that the role of biomarkers in our field today are really twofold. Number one is to make a more accurate diagnosis and help with kind of staging of progression. The other thing I think where our field is going, and I think our field will get there in the coming years and hopefully sooner, is that we can then use these blood-based biomarkers as an example to understand are the treatments that we're giving to patients, is the lifestyle behaviors that the person is making, is the person who's controlling their blood pressure and cholesterol and blood sugar versus not, are those people having different trajectories in terms of increasing amyloid or increasing tau or changes in neurodegeneration markers and shrinkage of the brain.

So I think what the field of Alzheimer's biomarkers is now is evolving quite rapidly. And whether you just do a spinal tap to look for amyloid and tau and look at the triplicate signature of different types of tau and amyloid, or you look at the blood plasma test, which there's only a few available currently in the United States amyloid-beta 42/40 ratio, as well as p-tau231 assessment is coming out soon, as well as other isoforms, including p-tau217 and p-tau181, and NfL [Neurofilament light chain] and GFAP [glial fibrillary acidic protein], which hopefully will be out in the coming years, or even sooner. Or even just doing an old-school FDG PET scan where you look at glucose metabolism. And if you have reduced glucose metabolism in areas of the brain that are kind of consistent with Alzheimer's disease that's another way to detect.

Basically, I think a lot of people look at Alzheimer's, as a homogeneous disease. But I really think that as we assess these biomarkers and track people long term over time, we'll understand that Alzheimer's is actually more of a heterogeneous disease, and different people may take different roads along that track.

Marc, what do you think about that?

Dr. Agronin:
I think this is a key takeaway, that we're gaining so much more information about what's going on in the brain. And then we really begin to appreciate, as you said, the variability in both clinical presentation and course. And that's essential in terms of knowing what we communicate to patients in terms of what's going on and what to do.

Well, this has been a great micro discussion. Unfortunately, time is up. Everyone, thank you for listening.

Announcer:
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