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When to Begin FcRn: Initiation Criteria, Key Evidence

Dr. Edmundson:
Today, we're going to dig into a very practical question. When do we move to FcRn therapy? Let's start by looking at what typically triggers this decision in real-world practice.

This is CE on ReachMD, and I'm Christyn Edmundson. Joining me today is Dr. Gil Wolfe.

So, Dr. Wolfe, when you're thinking about initiating FcRn therapy, what are the key clinical features that you look for, and how do you know that it's time to make that move to using an FcRn inhibitor to treat a patient with gMG?

Dr. Wolfe:
It's a great question, and we're still learning how to use these agents in the best way possible, but things to look for are these. You may have started a conventional therapy in a patient, but you're just not getting where you want to, say, within the first week or 2, and the patient is struggling.

So, the patient may fall into an MGFA clinical classification anywhere from 2, which is mild generalized disease, all the way up through 4B, which is severe generalized disease with a bulbar predominance, they could fall anywhere in there, but they're just not doing as well as you would like or they would like; that would be a patient to consider these agents in. We know the agents work quite quickly, which many of our conventional therapies don't, so that could be an added benefit of these newer agents.

If you are following the MG-ADL, which many of us do routinely in practice, if their score is hanging out above 5 or 6 with at least 3 of those points being non-ocular items, that could be a trigger. If you haven't been able to lower that MG-ADL down to a lower number, say, than 5 or 6.

If the patient is receptor or MuSK antibody positive, because those are the 2 forms of myasthenia gravis to this point that we have labeled indications for the neonatal FC receptor blockers, again, if they're uncontrolled with those serotypes, an FcRn blocker could definitely play a role in their treatment.

If there's high treatment burden, if they've been on long-term steroids at a fairly high dose with significant side effects such as hypertension, glucose intolerance, frequent needs for IVIG or plasma exchange as a rescue therapy or as a bridging therapy, that could be another avenue where you would consider an FcRn blocker.

An inability to taper immunosuppression, conventional immunosuppressive medications, you can't taper them because the patient's control suffers, that could be another reason to use it.

So, Dr. Edmundson, what do the trials tell us about who benefits, and how do you use that to guide conversations about starting FcRn

therapies?

Dr. Edmundson:
Yeah, great question. And of course, there's so much data coming from these clinical trials that I'm just really going to hit the high points here. So, currently there are 3 FDA-approved FcRn inhibitors for the treatment of generalized myasthenia gravis. Efgartigimod was the first approved, and that was studied in the pivotal ADAPT trial.

In the ADAPT trial, acetylcholine receptor antibody-positive patients were either treated with gMG or placebo for a cycle of 4 weekly infusions, and the efgartigimod-treated patients had a 67.7% MG-ADL responder rate compared with only 29.7% of patients treated with placebo. I will note that an MG-ADL responder was someone who had a 2-point or better improvement in their MG-ADL score for 4 consecutive weeks while they were being treated or in the observation period afterward.

The second available FcRn inhibitor is rozanolixizumab, which was studied in the MycarinG study that looked at both acetylcholine receptor antibody-positive and MuSK antibody-positive patients. The primary endpoint for that study was the change from baseline in MG-ADL score. The study looked at a low-dose drug, a high-dose drug, and a placebo arm.

In the low-dose drug, patients achieved on average a 3.37-point reduction in their MG-ADL score. The high-dose patients had a 3.4-point reduction in their MG-ADL score compared with the placebo patients who had only a 0.78-point reduction in their MG-ADL score.

In the MuSK subgroup, there were similar reductions in MG-ADL scores in the placebo versus drug-treated patients, although the numbers were much smaller.

The third and final FcRn inhibitor that's available on the market is nipocalimab. This was studied in the Vivacity-MG3 study, and it looked at patients with both acetylcholine receptor antibodies and MuSK antibodies. The primary endpoint in this study was the change in MG-ADL score from baseline in drug compared with placebo. In this study, the patients treated with nipocalimab achieved a 4.7-point reduction in their MG-ADL score compared with a 3.25-point reduction for the placebo-treated patients.

So, some key take-homes about this general class of drugs. The FcRn inhibitors tend to have a rapid onset of clinical effect. They're also targeted. They target a very specific part of the immune system. And finally, several of these drugs have outpatient administration options, which is nice for patients who don't want to go to infusion centers or who may even want to administer drug themselves.

Additionally, the use of FcRn inhibitors is often driven by a failure to achieve or maintain minimal symptom expression or an inability to de-escalate steroids in a patient's treatment regimen.

Patient selection should reflect trial inclusion logic but also allow for individualization for function, disease burden, and the overall trajectory of each patient's disease.

Well, this has been a great bite-sized discussion. Our time is up, and thanks so much for listening.