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

Facing Formidable Diabetic Cases with Patient Persistence & Adherence

Dr. Anderson:
Welcome to Diabetes Discourse on ReachMD. I'm Dr. John Anderson. And joining me to discuss a difficult case in diabetes and strategies to overcome it are actually two of my fellow hosts for the series, Dr. John Buse, and Dr. Carol Wysham. John, would you like to introduce yourself?

Dr. Buse:
Sure. My name is Dr. John Buse. I'm the director of the Diabetes Care Center as well as the chief of the Division of Endocrinology and Executive Associate Dean for clinical research at the University of North Carolina School of Medicine in Chapel Hill.

Dr. Anderson:
Thanks, John. And Carol, would you like to tell our audience just a little bit about yourself?

Dr. Wysham:
Yes. Hi. I'm Carol Wysham. I'm a clinical endocrinologist in Spokane, Washington who has a special interest in diabetes, 34 years of practice, and am the incoming President of the Endocrine Society.

Dr. Anderson:
Thanks, Carol, and sticking with you, I believe you actually have a patient to share with us today. Would you like to start by giving us some details?

Dr. Wysham:
Okay, so I've actually taken care of this fellow for pretty much his entire duration of his diabetes. He's 71, 25-year-history of type 2 diabetes. He's been relatively well controlled throughout his history. He's currently taking 1,000 milligrams of metformin, BID, he's on NPH insulin, he is taking 10 units in the morning, 60 units at night, and he's on regular insulin which he is supposedly counting carbohydrates and taking 1 unit per 5 grams of carbohydrate with each meal. He does his testing sporadically, generally one, maybe twice a day some days. His A1cs had been between 6.5 and 7.5 on this until June of this year, when he presented with an A1c of 8.5. He's got complication is only neuropathy. He has hyperlipidemia, hypertension, and sleep apnea. His examination was really just remarkable for his weight of 251 pounds. His blood pressure was a little high at 142/70 and he did have evidence of peripheral neuropathy and decreased peripheral pulses. His LDL was 65. His chemistry profile was otherwise unremarkable. His GFR was 60. And his albumin to creatinine ratio was 32.

So I'm faced with a fellow who has suddenly had a worsening of his control. Working with him, I think one of the important things that we need to learn how to do is to gently probe the adherence issue with him. And in doing so, I just asked him, so how many times a week are you missing your regular insulin? And his initial thought was, well, maybe, you know, once or twice a week and I said, Well you're still working, aren't you? Yeah. So do you take it when you go to work? Well, no. So now we've got five days a week, and so then after a bit, no, he doesn't take it when he goes out to eat and, and so now we find out he's actually missing more like 10 of his injections per week. So, that's how his initial history and physical ended, and I'd like to actually throw it back to you and to John to just discuss what you would do in this setting.

Dr. Anderson:
So, John, really getting to the bottom of persistence and adherence in our patients in a non-threatening way is really important because having diabetes, particularly if you're on bolus insulin, it's not very easy for patients.

Dr. Buse:
Absolutely, you know, I have to say, I tend not to use multiple daily injections in patients with type 2 diabetes predominantly for this reason. The way I enable people to talk about non-adherence is just to point out that taking multiple daily injections really involves taking 20 to 30 injections a week. And that's hard to do. I often relate that, I take a statin and I have to admit, I miss one at least every couple of weeks, just by mistake. So, it's okay to be imperfect in medication taking. But that communication is really the key so that I understand what they're doing.
You know, this is a really interesting case for two reasons. One with the GLP1 receptor agonist and the data that we have about switching people from multiple daily injections, we know he has a good shot of achieving adequate control with one shot of GLP1 receptor agonist a week and one shot of basal insulin a day or even one of the combined products. But we also have that little tickler that Carol gave us that the patient has a GFR of 60, and some albuminuria. That is not where the strongest evidence is for the use of SGLT2 inhibitors to prevent the progression of kidney disease. But it would be intriguing to think about using an SGLT2 inhibitor for this gentleman for the prevention of a progression of his kidney disease.

Dr. Anderson:
Right. I agree. So, Carol, after discussion with your patients, as y'all sort of negotiated next steps, what happened?

Dr. Wysham:
So I think John's points are very good in terms of not using basal bolus insulin therapy, certainly after they studies showing the equivalent if not better outcomes with comparing GLP1s to basal bolus insulin therapy. I certainly agree with John, I don't do that going forward. But an awful lot of my patients, I've been in practice here for almost 30 years, they're on basal bolus insulin therapy. And now I'm looking at backing them off. So this is a fellow who I discussed the risk, benefits, costs, and you know, what it would mean for him. And he agreed that he would like to try a GLP1. He had been on one in the past, by the way, and decided to stop it because of cost. So we had to certainly talk about that as well. And so what we chose to do is to start him on semaglutide. I stopped his regular insulin, I stopped his morning dose of NPH and lowered his evening dose to 50. And then asked him to do glucose testing as often as he would so that we could just make sure he didn't have a sudden rise in glucose, which he did not. And he came back after two weeks. Again, we did this by tele visit. And he reported feeling well, he was doing glucose testing two to three times a day. And most of his blood sugars were under 150. And he was only on the 0.25 mg of semaglutide. And so my question to you and to John would be: Would you continue to raise his dose again, being concerned about cost? Or would you be satisfied if he comes back in three months and his A1c is in the low 7s?

Dr. Anderson:
Well, I think if you want to talk about a target A1c for him, 7.0 to 7.5% for someone like him is probably completely reasonable. And if you can get there on a 0.25-mg dose, assuming you don't really want to achieve additional weight loss or that that's really a primary goal, because we know you're going to need to go up to the 0.5 or the 1-mg dose to see more weight reduction, then I think it's perfectly acceptable. I'll let John say what he thinks.

Dr. Buse:
Yeah, you know, I have to say I would probably try and reduce his insulin further, work on weight loss. And the background thought there would be the guy does have some evidence of kidney disease. Where the GLP1 receptor agonist have evidence with regards to kidney disease is in the prevention of progression towards macroalbuminuria, which is a strong marker for the risk for developing end-stage renal disease. So, particularly if you thought this guy had a long life expectancy, I might be inclined to push him forward, but at the same time, it'd be shared decision-making, and if cost is an issue, etc, etc. You know, he is doing fine, and you could continue at 0.25 mg.

Dr. Anderson:
For those just tuning in, you're listening to Diabetes Discourse on ReachMD. I'm Dr. John Anderson, and today, I'm speaking with Drs. John Buse and Carol Wysham, who's sharing a challenging patient case with us. So Carol, for this patient, and for patients like him, how do you have the conversation about the potential for GI side effects as you start a GLP1 receptor agonist?

Dr. Wysham:
Well, it just probably the same issue that he has probably about a one in five chance of having some nausea as we get started. And then if it does occur as we go up in dose, it generally does not last for more than a week or two. And certainly, he should make sure that he takes his dose according to schedule. And if he's still having some mild nausea at the end of the month that he can tolerate, to perhaps hold off on increasing the dose until his nausea is completely resolved. Turns out this guy had not a twinge of GI side effects, which, of course, in our experience, 80% of people don't. So that's the flip side. I like to tell people 80% of people don't have it. I did decide to go up. He was very excited about how he's doing. He'd already lost, believe it or not, 10 pounds in the course of a couple of weeks. Now, I think there's a backstory there, you know, suppressing the appetite. But also the excitement of seeing his blood sugars better, perhaps

feeling better. I think he was probably being a little better with his diet. But he was very excited. And this guy's an architect, his concern about money was he didn't like seeing the money go out of his pocketbook, he could afford the medication. So I lowered his dose of insulin by another 20% went up to 0.5. And we'll see him back in another month or so to decide where to go from there.

Dr. Anderson:
Sounds great. John, what do we know about GLP1 receptor agonists and their use in patients with chronic kidney disease?

Dr. Buse:
Well, not as much as we like. There's a big trial call FLOW that will specifically address this issue. But what we know for now is in many of the studies and in the meta-analyses of the cardiovascular outcome trials, there is a statistically significant reduction in the progression to macroalbuminuria. And, you know, less strong evidence about progression to doubling of serum creatinine and those kinds of more renal insufficiency kind of markers. How that happens is uncertain. There is some evidence that there are direct renal effects. It may also just be improvement in glycemic control, reduction in weight, reduction in blood pressure; all of which are risk factors for progressive kidney disease. So, bottom line, we don't know enough, but it does seem to have an effect. But it is important to point out that the effect of the SGLT2 inhibitors on progression of kidney disease is much better established.

Dr. Anderson:
Right. And, Carol, particularly for use of injectable semaglutide, you didn't have to worry about EGFR in this patient, did you?

Dr. Wysham:
No. In fact, you know, John's points about the kidney disease are really important. But with an isolated first-time ACR of 32, I was not really focusing on that because in order to make the diagnosis of microalbuminuria, or diabetic nephropathy, you really need to see the ACR being elevated on at least two occasions. So that was the first time I wasn't really focusing on that, although obviously, if it persists, we'll have that conversation about whether or not to look towards an SGLT2 inhibitor in addition.

Dr. Anderson:
Well, I think it's a great point. And I think this gentleman, was he a Medicare patient? Or does he still have private insurance?

Dr. Wysham:
Yes, he’s Medicare.

Dr. Anderson:
Yeah, you know, it's important to point out that I think a lot of providers just shied away from the use of GLP1 receptor agonists in Medicare patients for so long, that they don't realize that there's actually formulary coverage for some of these patients, that it's not as prohibitive as it used to be and to not exclude the possibility of something like an SGLT2 inhibitor or GLP1 receptor agonist, just because you have a Medicare patient.

Dr. Wysham:
And that's been my concern as well, I've had a hard time making that switch from not using them because of cost to now realizing their benefits, particularly in people who have had advanced to multiple daily injections of insulin.

Dr. Anderson:
And I think it's fascinating that having those conversations with your patients, getting to that non-threatening way of understanding adherence and what the patient wants and discussing preferences, making it a patient centered-decision is something we need to all be doing when we take care of any chronic disease, but particularly for those of our patients with diabetes.

Well, that’s all the time we have for today. But I want to thank you, Dr. Wysham, for walking us through this patient case, and you as well, Dr. Buse, for sharing your insights. It was great speaking with you both today!

Dr. Wysham:
Thank you, John. It was great to be here.

Dr. Buse:
Thank you.

Dr. Anderson:
That’s all the time I have for today. But to access this and other episodes from this series, visit reach-m-d-dot-com/DiabetesDiscourse, I’m Dr. John Anderson. Thanks for listening.