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Released: 08/31/2026
Valid until: 08/31/2027
Time needed to complete: 56m

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Translating Evidence into IgAN Care

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Dr. Barratt:
This is CE with GLC, and I'm Dr. Jonathan Barratt. Here with me is Dr. Jörg Latus, and in this episode we're going to review a recent patient case to illustrate how to apply recently updated KDIGO guidelines in clinical practice.

And Jörg, I understand that you have a patient case for us to discuss today.

Dr. Latus:
Thank you, Jonathan. Yes, I would like to start with a patient whom I saw in my clinic just this morning.

So this was a 20-year-old man, and he had a 1-year history of hypertension. He had been started on an angiotensin receptor blocker therapy by his GP 1 year ago, and after they started the therapy, the blood pressure was 120/80, so it was under good control. Apart from the hypertension, he had no relevant medical history and was not taking other medications.

Then there was a routine follow-up, and they tested the kidney function. And at this time, they measured a creatinine of 1.8 mg/dL, corresponding to an eGFR of approximately 50 mL/min. At this time, the patient was sent to my clinic.

At presentation, of course, I did urinalysis again, and kidney function remained unchanged, meaning the creatinine was still 1.8 mg/dL. I did the urinary analysis, and we saw microscopic hematuria and a proteinuria of 1.6 g/g. So, of course, given the young age of the patient, the impaired kidney function, and significant proteinuria and hematuria, we decided to go for a kidney biopsy.

So one day later, we did a kidney biopsy, and histology confirmed the diagnosis of IgA nephropathy. And the Oxford score was M1 E0 S1 T0 and C0. Based on this finding, of course, we talked to him and told him, "Yes, you are a high-risk patient." And at the time we did the kidney biopsy, we started a therapy with an SGLT2 inhibitor as an add-on therapy to his angiotensin receptor blockade.

At the follow-up visit, we of course discussed the histology results with him and of course addressed several options now available for him. And we started, of course, with maybe therapeutic options with Nefecon. But the patient was initially hesitant because of concerns regarding possible side effects of Nefecon.

Currently, of course, because proteinuria was still more than 1 gram, I put the patient on sparsentan, at the beginning 200 mg, and then I

increased the dose to 400 mg. And in addition—and maybe we can discuss this later on—I'm now evaluating him whether he may be eligible for enrollment in my study with the CD38 antibodies.

So I think this case illustrates several important findings. First, of course, we need a kidney biopsy in a patient to confirm the diagnosis of IgA nephropathy.

Of course, in patients with hypertension, we should do not only eGFR and creatinine; we should, of course, look for microhematuria and albuminuria. And then, of course, I believe the approach addressing both drivers of the disease are very important. So now he is on sparsentan and SGLT2, and maybe I'm going to include him in a study with CD38 antibody.

So, Jonathan, is this consistent with the type of patients you see in your practice, or what do you think? Am I doing it the right way, or what do you think?

Dr. Barratt:
Yeah, really sadly, this is typical of a patient I see. We need to work with our GPs to help them understand if you're 20 with hypertension, there is something seriously the matter, and please think about the kidneys because there was at least a 12-month period here where his kidneys were ignored, weren't they, Jörg? And it was only when they decided to check some blood tests, having been on antihypertensives, they suddenly realized that this was the kidneys driving the high blood pressure.

So yes, I see these late referrals. Sadly, this man in his 20s has already got significant kidney impairment. He's already lost a significant number of nephrons, and that disease is reflected in the degree of proteinuria that he has.

So I completely agree with you. A quick kidney biopsy and quick intervention, managing both the immune aspect of the disease and sadly having to address the CKD aspect because he has lost a significant number of nephrons.

And so what do we have in the UK? The same as in Europe. We have, from a disease-modification perspective, we have the gut-directed steroid Nefecon, and I would certainly consider using that in this gentleman, like you did. And then we have the drugs to manage the CKD aspect of the disease. And he was initially on a renin-angiotensin system blocker, and I completely agree with you. He was converted from that renin-angiotensin system inhibitor to sparsentan, the dual endothelin angiotensin receptor antagonist, and he was on an SGLT2 inhibitor, so exactly what I would have done.

And I again hear from people that they are concerned about taking Nefecon because they hear it's a steroid, and lots of people have heard lots of terrible stories about the side effects of systemic steroids, prednisolone, methylprednisolone.

Now, we know that Nefecon is not associated with as many of those side effects, but some patients still do get side effects. I'm not sure whether this young man tolerated the drug, but certainly if he didn't tolerate the Nefecon and I was giving him the sparsentan and the SGLT2 inhibitor and he remained, in my view, at risk of progressive loss of kidney function, then I would absolutely, like you, consider putting him into one of my open clinical trials. And all the clinical trials allow patients in on stable, optimized doses of sparsentan and SGLT2 inhibitors. He wouldn't be eligible if he was on Nefecon, however.

But I agree with you. This is a delayed diagnosis due to a delayed referral to you. We need to work better with our primary care colleagues to get that referral quickly, to raise awareness that in young people, being hypertensive has to be investigated. Quick biopsy, quick intervention using the drugs that we have available, and entirely consistent with the KDIGO guidelines.

Dr. Latus:
I think this is very important. We need the early diagnosis. And again, I'm going to discuss, of course, Nefecon again with this patient. But as you told us, we are not able to put them in a study. I think that's a disadvantage.

But again, it makes absolutely sense to address both drivers of the disease. So just after I put him on an SGLT2, I didn't measure again the proteinuria, but because maybe in that case I'm below 1 gram, that means I'm not able to prescribe the new drug.

So yes, we need an early diagnosis, and of course we have to address both drivers of the disease.

Dr. Barratt:
Yeah, and again, I'm not going to get tired saying this: One of the things we need to work really hard as nephrologists is working with our secondary care and primary care colleagues to raise awareness of glomerular disease, which is often silent but needs to be thought of, particularly in young patients presenting with hypertension, with borderline urine abnormalities, borderline creatinines, eGFRs, because we now have a way to intervene to protect these young people's kidneys with the wonderful new treatments that we're seeing. But they'll only work if we start them when there are nephrons left to save.

So for me, working with our colleagues to raise awareness of glomerular disease is important, and targeting both the immunological aspect of the disease and the CKD aspect of the disease simultaneously and optimizing that approach is key in this 20-year-old to give him the best chance of never seeing a dialysis machine in his lifetime.

So that's all our time. Thank you for your attention.

Dr. Latus:
Thank you very much.

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