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/monitoring-fsgs-traditional-and-novel-biomarkers/37187/

Released: 09/19/2025
Valid until: 09/19/2026
Time needed to complete: 1h 03m

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

Monitoring FSGS: Traditional and Novel Biomarkers

Announcer:
Welcome to CE on ReachMD. This activity is provided by Medtelligence and is part of our MinuteCE 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. Sise:
This is CE on ReachMD, and I'm Dr. Meghan Sise. Here with me is Dr. Vladimir Tesar. We're going to be discussing biomarkers that may be helpful when managing FSGS, including those that are going to help predict disease progression.

So traditionally, nephrologists have diagnosed FSGS with a kidney biopsy. The kidney biopsy also provides important prognostic information such as the type of FSGS, the degree of podocyte foot process effacement, and the degree of scarring. Repeat biopsies are uncommon in FSGS after diagnosis. Nephrologists usually just measure proteinuria, albuminuria, and assess the serum creatinine to calculate estimated glomerular filtration rate. And over time, we evaluate how the eGFR slope is declining.

There are novel biomarkers being assessed for FSGS. I'm going to start with genetic testing since it's available now in many parts of the world. The increasing use of genetic testing panels, which can test more than 60 podocyte-specific genes, have helped to understand the proportion of FSGS that's genetically mediated. And this can help us avoid unnecessary immunosuppression in patients that have genetically mediated FSGS.

APOL1 genotyping is becoming increasingly available and is really important in patients of recent African ancestry. This will help us identify patients who may qualify for APOL1-targeting clinical trials.

Emerging research has also suggested that there are additional promising candidates for phenotyping and monitoring FSGS, but none of the next tests I'm going to talk about are ready for clinical use right now. There are some research labs that can measure anti-nephrin antibodies. These may identify immune-mediated nephrotic syndromes and are more tightly linked with minimal change disease, but can be found in patients with FSGS as well.

There's also urinary exosomal micro RNAs, or plasma micro RNAs, that are shed from podocytes into the urine or can be detected in the plasma, and in the future, these may be used to monitor disease activity.

There are also proposed circulating factors such as suPAR or cardiolipin-like cytokine 1 that could be increasing glomerular permeability, and that may be a driver or podocyte injury. Again, these are not yet clinically available assays and the degree to which they predict progression is yet unknown.

There's also anti-CD40 antibodies. Again, not widely available, but may help subphenotype disease, particularly disease that's likely to relapse after a kidney transplant.

So, Dr. Tesar, what are your thoughts about the role of biomarkers in the diagnosis, treatment, and monitoring of FSGS?

Dr. Tesar:
You covered very nicely the main areas probably of future development, because currently, we still have in hand mostly the protein-creatinine ratio. But of course, proteinuria, or protein-creatinine ratio, may not be able to diagnose early disease to quantify small changes in the damage to the kidney, not to try to distinguish between damage and activity of the disease. So we would need new ones. Probably anti-nephrin antibodies and other antibodies. So I think that currently we must stay with proteinuria, and also I would promote genetic testing as well suggested for the future.

Of course, there may be some other options, such as single-cell transcriptomics in kidney biopsy or using artificial intelligence for evaluating the kidney biopsy. But there is a very limited experience with these methods in FSGS.

And so we stay with proteinuria, which is reflecting well the damage and the outcome of the patients. And we also know that this is a modifiable parameter, and any decrease of proteinuria is translated into a good long-term outcome of the patient, and this is very important in terms of finding new medications and having some surrogate endpoint.

Dr. Sise:
So I agree. I think in the coming years, we will understand proteinuria better. And some of those insights will come from PARASOL and other groups that are working hard to determine how we interpret proteinuria in light of new therapies that lead to proteinuria reduction. And I also think in the coming years, we will see validation of novel biomarkers that help us subphenotype FSGS and I think these biomarkers will eventually be incorporated in clinical decision-making algorithms.

So this has been a great discussion. Thank you, Dr. Tesar, for joining me and thanks to the audience for listening.

Announcer:
You have been listening to CE on ReachMD. This activity is provided by Medtelligence and is part of our MinuteCE curriculum.

To receive your free CE credit, or to download this activity, go to ReachMD.com/CME. Thank you for listening.