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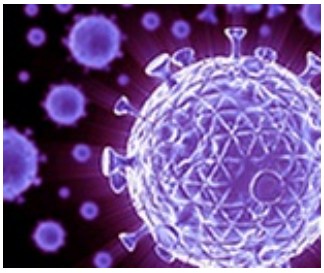

VOLUME 3 - ISSUE 6

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New Advances in ART

- Recognize risk factors for the development of chronic kidney disease in patients on ART.
- Summarize the benefits, risks and rationale for switching from tenofovir disoproxil fumarate (TDF) to tenofovir alafenamide (TAF) in well-controlled and treatment-experienced patients.
- Discuss novel long acting therapies for HIV.

Guest Faculty Disclosure
Dr. Simmons has disclosed that she has received honoraria from the American Board of Internal Medicine, BMJ, and DynaMed. Also, that her immediate family has stock options with Eli Lilly and Company.

Unlabeled/Unapproved Uses
Dr. Simmons has further indicated that, except as noted specifically within the discussion, there will be no references to the unlabeled or unapproved uses of any drugs or products in today's program.

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Podcast Transcript

BOB BUSKER: Welcome to this *e*HIV Review podcast.

I'm Bob Busker, managing editor of *e*HIV Review. Our guest today is Dr. Rachel Simmons, Assistant Professor of Medicine at Boston University School of Medicine. And our topic — a follow-up to Dr. Simmons's recent newsletter issue — is New Advances in ART.

***e*HIV Review is jointly presented by the Johns Hopkins University School of Medicine and the Institute for Johns Hopkins Nursing. This program is supported by educational grants from Gilead Sciences, Inc. and ViiV Healthcare.**

Learning objectives for this audio program include:

- **Recognize risk factors for the development of chronic kidney disease in patients on ART.**
- **Summarize the benefits, risks and rationale for switching from tenofovir disoproxil fumarate (TDF) to tenofovir alafenamide (TAF) in well-controlled and treatment-experienced patients.**
- **Discuss novel long acting therapies for HIV.**

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Dr. Rachel Simmons, thank you for joining us today.

DR. RACHEL SIMMONS: Thank you, I'm thrilled to be here.

MR. BUSKER: In your newsletter issue, Dr. Simmons, you reviewed the results of recent studies that may improve antiretroviral therapy for a number of HIV-infected patient subgroups. Today I'd like to translate some of that information into clinical practice. So please start with a patient scenario.

DR. SIMMONS: Our first patient is a 50-year-old man with HIV on tenofovir disoproxil fumarate (or TDF)/emtricitabine plus boosted atazanavir who presents for HIV follow-up. He would like to reduce the number of pills he takes to one if possible. His brother also recently developed kidney problems related to diabetes, and the patient would like to know about his risk factors for kidney problems.

His medical history includes HIV, his exposure was remote injection drug use. His HIV was diagnosed about 11 years ago when he presented with Pneumocystis pneumonia and his CD4 count was 67. His other medical condition is hypertension. His medications are TDF/emtricitabine plus ritonavir-boosted atazanavir and lisinopril. He quit smoking last year. His physical exam is notable for a blood pressure of 122/78 and is otherwise normal. Labs include a creatinine of 1.1, an estimated GFR of 74 mL/min, and no proteinuria on urinalysis.

MR. BUSKER: His concern about his potential for developing kidney disease — how would you estimate his risk?

DR. SIMMONS: We know that patients who are HIV positive have a higher risk of developing chronic kidney disease than the general population, and that's probably due to a number of factors, related to both the virus and to medication.

A group looked at a longitudinal cohort study of predictors of risk for chronic kidney disease and they came up with several things. They used the cohort study, the Data collection on Adverse events of Anti-HIV Drugs, (D.A.D.) Study, which is a large, prospective study of HIV positive patients in Europe, the United States and Australia. In their cohort, they looked at almost 18,000 individuals with more than three GFR measurements who had had normal renal function at enrollment with an estimated GFR of > 60 mL/min. Those patients also had no history of tenofovir disoproxil fumarate use, atazanavir, or any boosted PI use prior to enrollment in this cohort. That's a little different than our patient, but I think their results are quite helpful.

In the cohort that they looked at, 73% of the patients were men, and race was unknown for about 44% of them. Several non-HIV and HIV related characteristics were noted to be significant predictors of chronic kidney disease. The first was increasing age, followed by female gender, hepatitis C coinfection, prior cardiovascular disease, lower eGFR at baseline, hypertension, diabetes, and intravenous drug use as compared to other HIV exposure groups. Lastly, they noted a lower nadir CD4 count < 200.

The study couldn't comment on the known association of race to risk for chronic kidney disease because race was unknown in such a significant proportion of patients. This database also contained no information about proteinuria, so couldn't comment about that as a risk factor for chronic kidney disease. The risk factors they identified were also similar to those discussed in the clinical practice guidelines for the management of chronic kidney disease in patients with HIV published in 2014 by Lucas and colleagues.

MR. BUSKER: The recent investigation using the data from the D.A.D. study — was that Mocroft et al, which you reviewed in your newsletter issue?

DR. SIMMONS: That's correct.

MR. BUSKER: And they developed a clinical tool for assessing the risk of developing chronic kidney disease. Tell us more about that.

DR. SIMMONS: Using this cohort, the authors then went on to develop a clinical risk tool to predict the possibility of developing chronic kidney disease in the next five years. The risk tool gave points for patient age, female gender, intravenous drug use, HIV exposure group, hepatitis C coinfection, a baseline GFR of > 70, nadir CD4 count < 200, and a history of hypertension or cardiovascular disease or diabetes. Using this risk model, individuals were determined to have either low, medium, or high risk of developing CKD. The chance of developing CKD over the next five years went up quite significantly based on risk group. The risk of developing CKD was about one in 400 in the low risk group and went up to one in six in the high-risk group.

The investigators then went on to look at the effect of starting different antiretroviral agents in the different risk groups, and they found that starting TDF or ritonavir-boosted atazanavir, or any other boosted protease inhibitor aside from lopinavir/ritonavir, was equivalent to increasing the risk score by about two points. That led the authors to determine the number needed to harm or the number needed to be started on a particular antiretroviral medication for one additional person to get chronic kidney disease over the next five years. They found that in the highest risk group, the number needed to harm was nine when starting a medication like TDF or atazanavir with ritonavir. Fortunately, this tool is available online for practitioners to use for their own patients. If we apply the clinical risk predictor to the patient we're discussing, it's estimated that his risk of CKD would be about 7% over the next five years.

This patient is already on tenofovir disoproxil fumarate and ritonavir boosted-atazanavir, and each of those would increase the risk to about 11% over the next five years. Mocroft and colleagues did an additional separate analysis where they looked at individuals in the D.A.D. cohort with normal renal function at baseline. They noted a cumulative risk of CKD with years of exposure to TDF or ritonavir-boosted atazanavir. In their multivariate analysis they noted approximately a two-fold increase in chronic kidney disease after five years of exposure to either TDF or ritonavir-boosted atazanavir. This is important information that I would share with this patient, given his concern about chronic kidney disease.

MR. BUSKER: Beyond sharing that information with the patient, how would you monitor his actual renal function? What do the recommendations say?

DR. SIMMONS: For all patients who are infected with HIV, it's recommended that they have regular monitoring of their renal function. That means if they have a basic chemistry where you check the creatinine and can estimate the GFR at enrollment into care when antiretroviral therapy is initiated or modified, then at least every six months when patients are on a stable regimen.

The other parameter that we follow is a urinalysis. Urinalysis is recommended again at entry into care and then at least annually, and more often for patients who are at risk of renal disease. So for patients who are on either tenofovir alafenamide, or TAF, or TDF, urinalysis is recommended every six months. Some experts also recommend following quantitation of either albumin or protein in the urine by checking microalbuminuria or protein-to-creatinine ratios. Certainly, if patients have evidence of proteinuria on urinalysis or urine dipstick, it's recommended to quantify that further with either a microalbuminuria or a urine protein-to-creatinine ratio.

Given our patient's risk factors for chronic kidney disease and his current antiretroviral regimen, I would recommend checking an estimated GFR at least every six months with the MDRD equation, and I would also check a urinalysis every six months and measure either urine protein-to-creatinine ratio or a microalbumin. I would also work with him to make sure we're addressing other modifiable risk factors for kidney disease, including making sure we're treating his hypertension to goal, congratulating him on his smoking cessation, and avoiding additional nephrotoxic agents such as long-term NSAID use.

MR. BUSKER: This patient, as you presented him, is also looking to reduce his pill burden. There are single tablet regimens available — which would you recommend for this patient?

DR. SIMMONS: Among the currently available single tablet regimens, I would consider for this patient tenofovir alafenamide, emtricitabine and elvitegravir with cobicistat. The newer single tablet regimens have swapped out tenofovir alafenamide. Tenofovir alafenamide is a novel prodrug associated with lower plasma levels and it has a better safety profile than TDF, and we'll talk about that in the next bit.

When we think about which medications of these six single tablets to include, each has important factors to consider such as food requirements and drug-drug interactions. Also single tablet regimens containing rilpivirine aren't recommended for patients with a viral load over 100,000 or a CD4 count < 200. In addition, certain drug-drug interactions to think about, for example. Rilpivirine is not recommended to be given with proton pump inhibitors like omeprazole, and in patients in whom

you're considering abacavir they must be tested for an HLA-B5701 and that must be negative prior to use.

MR. BUSKER: Switching to a single tablet regimen containing TAF, even for patients stable on their current regimens — that was the focus of the Mills study that you reviewed in your newsletter Issue. Please briefly review that for us.

DR. SIMMONS: Mills and colleagues conducted a randomized control trial for patients who are well controlled on their current regimen to switch to a single tablet regimen containing elvitegravir, cobicistat, emtricitabine, and TAF. They recruited about 1,400 patients from a number of Gilead trials of TDF-containing regimens. Those participants had an estimated GFR > 50 and they'd had viral loads of < 50 copies on their current regimen for at least 96 weeks. Mills and colleagues looked at virological control, which they defined as a viral load of < 50 copies at 48 weeks.

Just over 950 patients were switched to the single tablet regimen and 97% achieved a viral load of < 50 copies at 48 weeks, compared to 93% in the TDF continuation group. These results indicate that the group that switched to TAF was noninferior to continue the TDF containing regimen. In the switch to the single tablet regimen containing TAF, there were better bone and renal biomarkers.

The mean bone mineral density of the spine and hip increased from baseline in the TAF group, while the bone density decreased or stayed the same in the TDF group. In addition, the GFR in the TAF group increased while in the TDF group the GFR either decreased or stayed the same. Proteinuria was also significantly less in the TAF group. And both regimens were well tolerated, whether switched to the TAF containing single tablet or continued on the TDF regimen. The follow-up was on the order of 48 weeks, and they did not see any clinical outcomes of fragility fractures or proximal tubulopathy. This study can help us discuss with the patient the opportunity to switch to a single tablet regimen.

MR. BUSKER: Is switching from TDF to TAF something you'd recommend for the patient you presented?

DR. SIMMONS: I would discuss these results with the patient and suggest we switch to the single tablet regimen containing elvitegravir, cobicistat, emtricitabine, and TAF. He would be on fewer tablets, as he desires, and potentially would have lower risk of kidney issues. But clinical endpoints such as chronic kidney disease and fragility fractures are rare events, so we'll need additional longitudinal observational studies to evaluate these outcomes in patients who switch to TAF-based regimens.

MR. BUSKER: Thank you for that case and discussion, doctor. And we'll return — with Dr. Rachel Simmons from Boston University School of Medicine — in just a moment.

MR. BOB BUSKER: You've been listening to *e*HIV Review, a combination newsletter and podcast program delivered via email to subscribers.

Newsletters are published every other month. In each issue, an expert author reviews the current literature in an area of specific importance to clinicians treating patients with HIV, including infectious disease specialists, primary care physicians, nurse practitioners, physician assistants, and others.

In the month following each newsletter, the expert author provides a case-based podcast discussion like the one you're listening to now to help translate that new information into clinical practice. These podcasts are also available as downloadable transcripts.

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Thank you.

MR. BUSKER: Welcome back to this *e*HIV Review podcast. We've been talking with Dr. Rachel Simmons from Boston University School of Medicine about recent research that may improve clinicians' ability to more effectively and more safely manage their patients with HIV infection. So to continue in that clinical vein, please bring us another patient scenario.

DR. SIMMONS: Our patient is a 37-year-old HIV positive woman who is a single mother with two children and working full time who has struggled with medication adherence. While being treated with efavirenz, TDF, emtricitabine, she developed reverse transcriptase mutations K65R, M184V, and K103N. She started going to a local support group for HIV positive women and optimized her system for taking medications, and her adherence has greatly improved.

She is currently on etravirine twice daily, darunavir/ritonavir twice daily, and raltegravir twice daily. Her viral load has been < 20 copies/mL for six months. Her medical history is HIV, her exposure was heterosexual sex, and her HIV was diagnosed at a prenatal visit. Her medications are the etravirine twice daily, darunavir/ritonavir twice daily, and raltegravir twice daily. Her

physical examination is normal and her labs include a creatinine of 0.8 and an estimated GFR of 88 mL/min.

MR. BUSKER: How would you interpret her resistance mutations?

DR. SIMMONS: When she was failing her initial regimen, her HIV genotype showed the K103N, the K65R, and the M184V mutations. The first letter is generally the code for the amino acid in what we would consider a wild type HIV virus, the number identifies the position of that codon, and the second letter identifies what that amino acid has changed to in the new sample. With certain changes in the HIV virus, they can confer significant resistance to different HIV medications. Some helpful resources for interpreting HIV genotypes are the Stanford University HIV Drug Resistance Database, where you can plug in a patient's individual drug resistance mutations and get an interpretation, and the IAS-USA Drug Resistance Mutations Group also compiles and publishes a list of mutations associated with clinical resistance. These two references can be very helpful in interpreting an HIV genotype.

In this patient, the M184V mutation that causes high level resistance to the emtricitabine component of her regimen, the K65R mutation, confers intermediate to high level resistance to the TDF component, and K103N is the drug resistance mutation that confers high level resistance to NNRTIs, particularly efavirenz.

What unfortunately happened with this patient is that she developed resistance to all three elements of her initial antiretroviral regimen; therefore, she's on a more complicated regimen that involves four tablets twice a day.

MR BUSKER: So that's how her regimen got so complicated. What can you do now to simplify it?

DR. SIMMONS: Fortunately, we can evaluate a switch study that might give us an option for a simpler regimen. Huhn and colleagues conducted an open label, multicenter, noninferiority trial of switching elvitegravir, cobicistat, emtricitabine and TDF plus darunavir — two tablets —vs continuing current antiretroviral therapy in patients who were well controlled on their regimen but who had significant resistance in their background.

The participants were virologically suppressed for at least four months on a darunavir-containing regimen, and they had previously failed at least two regimens with a resistance to two or more classes of antiretrovirals. These were fairly significantly treatment-experienced patients and they were allowed to have certain resistance mutations on their genotype.

On prior genotypes, the resistance mutations that were permitted included a K65R, and up to three thymidine analog mutations. Several resistance mutations, though, excluded people from the study. Patients could not have any integrase strand transfer inhibitor mutations, and they could not have a Q151M mutation or a T69 insertion mutation or any darunavir-associated mutation.

They randomized 89 participants to the combination of the single tablet plus darunavir and had 46 patients continue their current regimen. The study population was roughly three-quarters men, half of the participants were white, and at baseline the median number of tablets per day was five. The most common mutations were similar to our patient and the two most common mutations included an M184V and a K103N. At 24 weeks they found that a significant number of patients had good virological control; 96.6% of patients in the switch group had virological control and 91% of patients in the continuation group had maintained virological control, thus indicating that the switch group on the simpler regimen was noninferior to continuing the current antiretroviral therapy.

When they looked at 48 weeks, the group on elvitegravir, cobicistat, emtricitabine, and TAF plus darunavir again met the predetermined criteria for noninferiority; in fact, at that point they reached the criteria for superiority, with 94% in the switch group achieving virological success compared to 76% in the continuation group achieving virological success. Fortunately, in the switch group no one developed confirmed virologic rebound or had to discontinue the therapy due to adverse events. This simplified regimen is one that I would consider switching her to and would discuss with the patient.

MR. BUSKER: What about the safety profile of this simplified regimen?

DR. SIMMONS: This newer regimen was well tolerated. They also looked at the change in eGFR during this study, and it was similar in both groups; however, the switch group had a significant decline in the level of proteinuria compared to the continuation group. Similarly to the study we discussed where patients were placed on a TAF-based regimen, the level of proteinuria went down.

MR. BUSKER: How did that simplified TAF-based regimen affect patient satisfaction?

DR. SIMMONS: The switch group had significantly higher mean satisfaction and there were fewer missed doses. I think this study highlights that simplifying complex regimens to newer fixed dose combination tablets plus a protease inhibitor is safe and effective in certain people whose HIV is well controlled on their salvage regimen. For our patient this would mean she's initially on four tablets twice a day, and if we switch to the regimen described in the study, it would reduce her tablets to two once daily.

MR. BUSKER: Thank you for that case and discussion. I think we've got time for one more patient scenario.

DR. SIMMONS: Our patient is a 25-year-old man with HIV on TDF/emtricitabine plus dolutegravir started three years ago. He's been doing well on his medication, though he expresses frustration with taking medications every day and needing to do so indefinitely. His medical history is notable for HIV and his physical examination is normal. His labs include a creatinine of 1.1 and an estimated GFR of 92 mL/minute. His HIV RNA has been < 50 copies/mL for over a year.

MR. BUSKER: Here's a patient who's doing well on his current regimen. Would you consider changing his meds to include TAF? And why might you do that?

DR. SIMMONS: I would consider changing this patient's regimen, and we have a recent open label switch study to help us think about that. Gallant and colleagues evaluated substituting TDF to TAF while maintaining the rest of an HIV treatment regimen in patients who are doing well on their current antiretroviral therapy.

In this noninferiority trial, 668 patients were randomized to continue TDF or to switch to a TAF-based regimen while remaining on their remaining component. All of the participants had a viral load of < 50 copies/ mL for at least six months prior to entering the study, and at 48 weeks they found that the TAF group had similar rates of virological success compared to the TDF group, with 94% and 93% with an HIV RNA of < 50 in the switch group vs the continuation group, respectively.

There were some upsides to the switch. The bone mineral density increased from baseline in the TAF group, but it decreased or remained the same in the TDF group, and the participants in the TAF group had a small but statistically significant improvement in GFR. They also had a reduction in the amount of proteinuria compared to the TDF group. Like the similar switch study we discussed earlier, both drugs were well tolerated, and the rates of adverse events were similar.

This study highlights that switching from TDF to TAF, both are similarly efficacious and switching doesn't compromise the high level of virological control that can be achieved with TDF. So I would discuss switching with our patient.

MR. BUSKER: There were upsides shown to switching at 48 weeks. Have any studies followed switch patients for a longer period?

DR. SIMMONS: Yes, they've published longer term follow-up at 96 weeks, which also showed that the TAF regimen was noninferior to the TDF regimen.

MR. BUSKER: So for potential benefits in bone density and kidney function, you would consider switching TDF to TAF. Are there any situations where you would not switch from TDF to TAF?

DR. SIMMONS: The group of drugs called rifamycins can potentially reduce the level of TAF, so coadministration is not recommended. For example, if a patient needed to take rifampin for a mycobacterial infection in addition to being treated for HIV, I would not recommend using TAF in that situation. Also, coadministration of TAF with tipranavir is not recommended. The HIV guidelines, as well as many pharmacy resources, have this helpful information about drug-drug interactions. In addition, at this time there is limited data about using TAF in pregnancy.

MR. BUSKER: The patient you presented, like many if not most patients with chronic conditions, wants to get way from the hassle of daily dosing. Talk to us about potential newer treatments that would eliminate the need for a daily tablet?

DR. SIMMONS: The results of the LATTE-2 trial were recently published which was a noninferiority trial of using two long-term injectable medications as maintenance therapy for HIV. Margolis and colleagues conducted a study with two novel, long acting antiretrovirals, cabotegravir and rilpivirine. Cabotegravir is an integrase strand transfer inhibitor and is an analog to dolutegravir.

The study involved treatment naïve adults with CD4 counts > 200. They were treated with an initiation phase of oral abacavir and lamivudine and cabotegravir for about 20 weeks, and if they maintained virological control, they were transitioned to either intramuscular injections every four or eight weeks of long acting cabotegravir and long acting rilpivirine, or they continued on the oral regimen.

The study population was 92% men and 15% were black. The average baseline viral load was about 4.4 log copies/mL, and roughly 18% had viral loads exceeding 100,000 copies/mL. Two hundred thirty participants were randomized to the long acting injectable medications every four weeks or every eight weeks, and 56 were randomized to continuing their oral medications. At 32 weeks the proportion of patients with an HIV viral load of < 50 copies/mL was 94% in the four-week injectable group and 95% in the eight-week injectable group, and 91% in the oral treatment group. So there was no significant difference in virological control among the groups, indicating that the long acting injectables were noninferior to continuing the oral regimen. In fact, at 96 weeks they also found that the regimens were similar in the rates of virological success.

DR. SIMMONS: All three treatment groups reported a high degree of satisfaction with their treatment regimens. However, when asked if they were highly satisfied to continue their regimen, 99% of patients in both the long acting regimens said

yes, and only 78% in the oral therapy group said they were highly satisfied to continue.

This study is a very intriguing phase 2 study that raises the possibility of using long acting injectable medications for HIV maintenance therapy. These formulations are not FDA approved, and additional treatment studies are ongoing at this time, including a phase 3 study of looking at giving this injectable long acting regimen for treatment of HIV patients who are treatment naïve and a phase 3 trial of switching to long acting cabotegravir, long acting rilpivirine from a variety of three drug regimens. So I would suggest to our listeners and to this patient to stay tuned.

MR. BUSKER: Thank you for your insights into today's cases. Let's wrap things up by reviewing our discussion in light of our learning objectives. To begin: recognizing the risk factors for developing chronic kidney disease in patients on ART.

DR. SIMMONS: Risk factors include HIV exposure risk group of intravenous drug use, hepatitis C coinfection, increasing age, female gender, a low nadir CD4 count of < 200; and other coexisting conditions including hypertension, cardiovascular disease, and diabetes. And even though this was not addressed in the study we discussed, African-American race or African descent has also been shown to be a risk factor for chronic kidney disease in people with HIV.

There was also a clinical risk tool that was developed to estimate the risk of CKD over five years. This tool is available online and you can plug in the information for your own patients to customize your care.

MR. BUSKER: And our second learning objective: the benefits, risks, and rationale for switching from TDF to TAF in well-controlled and treatment-experienced patients.

DR. SIMMONS: TAF is a newer prodrug of tenofovir with lower plasma levels, and it seems to have less effect on bones and kidneys. Patients who switch from a regimen containing TDF to TAF experience similar excellent virological control and seem to have better bone and renal parameters. A second study we discussed also showed that in patients who are highly treatment-experienced with certain drug mutations, they can be transitioned to a simpler regimen containing TAF.

I would not recommend switching to TAF if the patient needs to take a rifamycin for a second type of infection. In addition, limited data is available for TAF in pregnancy.

MR. BUSKER: And our final learning objective: novel long acting therapies for HIV.

DR. SIMMONS: A recent study of long acting therapies for HIV is intriguing. This study looked at giving cabotegravir and rilpivirine by injection either every four weeks or eight weeks. They found that patients were able to maintain excellent virological success for maintenance therapy with this regimen. The people in the study expressed a high degree of satisfaction on this long acting injectable regimen. We will all have to stay tuned for future results regarding these long acting therapies.

MR. BUSKER: Dr. Rachel Simmons from Boston University School of Medicine, thank you for participating in this *e*HIV Review Podcast.

DR. SIMMONS: It was a great pleasure, thank you very much.

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