

Expert Interviews

National HIV Curriculum Podcast

Evaluation and Management of Isolated Hepatitis B Core Antibody for Persons with HIV

July 30, 2026

Season 3, Episode 5

In this National HIV Curriculum podcast episode, Dr. Brian Wood, Editor for the podcast series, interviews Dr. Kim, an expert in viral hepatitis, to discuss the evaluation and management of isolated hepatitis B core positivity for people with HIV.

Topics:

- Hepatitis B
- Immunizations
- Isolated core antibody
- switching ART

Brian R. Wood, MD
Professor of Medicine

Division of Allergy & Infectious Diseases
University of Washington

[Disclosures](#)

Disclosures for Brian R. Wood, MD

None

H. Nina Kim, MD, MSc

Professor of Medicine
Division of Allergy & Infectious Diseases
University of Washington

[Disclosures](#)

Disclosures for H. Nina Kim, MD, MSc

None

References

[Paper #1](#)

Transcript

Read along with the audio or jump to a particular chapter.

In this episode:

- [Introduction](#)
- [Isolated Hepatitis B Core Defined](#)
- [Why We See Isolated Hepatitis B Core](#)
- [Most Common Scenario in HIV Care](#)
- [Considering Hepatitis B Vaccination](#)
- [Vaccines and Isolated Core Antibody](#)
- [“Boosting Immunity”](#)
- [Switching off Tenofovir: What now?](#)
- [Take-Home Messages and Closing](#)
- [Credits](#)

[introduction](#)**[00:00] Introduction**

Hello everyone. I'm Dr. Brian Wood from the University of Washington in Seattle. Welcome to the *National HIV Curriculum* podcast. This podcast is intended for health care professionals who are interested in learning more about the diagnosis, management, and prevention of HIV.

Well, I would like to welcome Dr. Nina Kim. Dr. Kim is professor of medicine in the Division of Allergy and Infectious Diseases here at University of Washington. She's also adjunct professor in the School of Public Health, specifically in Health Services and Population Health. She's a provider here at our Madison Clinic, the Ryan White-funded HIV clinic, and also provides care at our hepatitis and liver clinic. She has led a number of studies related to viral hepatitis in people with HIV. She also serves on the OI [Opportunistic Infection] Guidelines Panel, specifically for management of hepatitis C, and has a lot of expertise in hepatitis B as well. She's also been a mentor of mine for many years. Nina, welcome.

Dr. Kim

Thanks, Brian. I'm very glad to be here. And to dive into this topic of isolated core, which I think is easily one of the most common questions I get as a hepatitis expert.

Dr. Wood

It's common, and it's confusing. So, I'm glad we're diving into this. I think a lot of learners and clinicians hear isolated hep B core, and what it means can be a little perplexing. What to do with it can certainly be confusing. So, maybe Nina, we can just start with some fundamentals.

[isolated-hepatitis-b-core-defined](#)**[01:30] Isolated Hepatitis B Core Defined**

Dr. Wood

If I'm seeing someone in HIV clinical practice and I send a whole hepatitis B serology panel, what do we really mean when we say isolated hep B core?

Dr. Kim

When you're screening anybody for hepatitis B, you want to send the full panel, and that's also been referred to as "triple screen," which is basically that has three components in the testing, which is the core antibody, surface antibody, and surface antigen. And essentially, the isolated core profile is someone who is positive just for hepatitis B core antibody and not for either of the surface antibody or surface antigen.

Dr. Wood

And so how do you interpret that differently if someone has a positive core antibody and negative surface antibody versus if they have a positive core antibody and a positive surface antibody?

Dr. Kim

Let me step back a little bit and say a few words about what the core antibody represents. Basically, it reflects the fact that the individual has likely seen the actual virus at some point in their lives. So, an immunized individual or someone who's never been exposed to natural infection wouldn't have core antibody. So, for example, an immunized individual would develop surface antibody in the absence of core antibody. The other point I wanted to bring up is that clinicians tend to confuse core antibody with core antigen, which is actually a viral protein found in the core of the hep B virus (HBV), and are sometimes under the impression that core antibody means they have active infection, but that is not necessarily so. Most of the time, you have resolved infection. The thing about core antibodies is that we can't assume immune protection with core antibody positivity.

Dr. Wood

And I think we'll come back to that, why we can't assume immunity specifically for people with HIV, but let's just pause a little bit more here on the basics and the fundamentals: So, hepatitis B core antibody means someone has been exposed to the virus in the past, and an isolated core means that's all you're seeing that's positive, surface antibody-negative, surface antigen-negative. How often do you see that in your clinical practice?

Dr. Kim

Well, it depends on the patient population you're looking at. So, for people with HIV, you could see this anywhere; most case series is somewhere in the ballpark of 10 to 20%. I think it's about 15% in CNICS [[Center for AIDS Research Network of Integrated Clinical Systems](#)], for example, which is, kind of, the Consortium of Centers for AIDS Research sites that I'm most familiar with. But you can see this profile in up to 40% of people with HIV in some case series. And just to put it in some contrast to less than 1% seen in general population studies. So, most people out there have not been exposed to hepatitis B, but people with HIV, there's specific risk factors related to having HIV that also put you in the way of having seen hepatitis B before. So yeah, it's a pretty common profile.

Dr. Wood

So, providers will see this in HIV clinical practice. Anyone who's doing HIV care, HIV primary care, will see this isolated hep B core antibody pattern, 10 to 20%, maybe even up to 40%. So, 1 in 10, 1 in 5 individuals may have this pattern of serology results.

[why-we-see-isolated-hepatitis-b-core](#)**[04:51] Why We See Isolated Hepatitis B Core**

So, let me then turn to asking you, given this is so common, what are the possible explanations? What comes to your mind in terms of why we might be seeing this?

Dr. Kim

So, there're one of four possible scenarios. I'm going to talk about two rarer scenarios that we could sort of just set aside for the moment, which is the window phase, which is a period of time when someone is newly infected when the surface antigen has gotten to below detectable levels, but the surface antibody has not yet quite developed. And in that seronegative window, you can sometimes just get an isolated core antibody. But keep in mind that you're not going to encounter this that often. It's a very discreet transient phase in an individual who likely otherwise would have clinically apparent manifestations of acute hepatitis B, including very high liver aminotransferases, ALT [alanine aminotransferase], and AST [aspartate aminotransferase] in the 1000s range.

Another scenario is a false-positive core antibody. So, these individuals would actually be susceptible to hep B, but this too is thought not to be very common, particularly with our current assays, but that is definitely a possibility as well. I think when you're looking at people with HIV, for example, the probably more common scenario is that someone has had hepatitis B, they can encounter the virus sometime in their lives but went on to resolve their infection. At some point, did have a surface antibody, but they basically had waning of their surface antibody to a level that's less than 10 or 12 international units per liter (IU/L), which is, depending on the assay, the threshold that we consider protective.

The final scenario is someone who had chronic infection. So, they had surface antigen at one point, so they could have this either through developing increased immunity and eventually losing the surface antigen, which is by definition our definition of a functional cure—that is, sustained loss of surface antigen that can happen with or without surface antibody later developing. But these folks can sometimes just show up as an isolated core.

And then they're going to be people who are... This is kind of a subset of that category who actually develop escape mutations—escape or diagnostic. So, both immune or diagnostic mutations that result in low production of surface antigen. So, if they're not on HBV-active ART [antiretroviral therapy], they're going to have evidence of HBV viremia. In a lot of cases, these are the folks who have occult hepatitis B.

Dr. Wood

So, if I'm understanding you correctly: so, the unlikely scenarios will be false-positive hepatitis B core or an acute window phase, but then there are two other possibilities: Someone has been exposed to the virus and developed immunity, but the surface antibody level has waned, and we're just not picking it up; or chronic infection, occult hepatitis B, and we're not detecting the surface antigen for various reasons, escape mutations or other reasons. Am I understanding that correctly?

Dr. Kim

That's right.

[most-common-scenario-hiv-care](#)**[07:58] Most Common Scenario in HIV Care**

Dr. Wood

Which of those scenarios is most common in HIV clinic for people in primary care HIV practice? Which of those explanations should we think of as most likely?

Dr. Kim

The most common scenario is likely to be waning of the surface antibody. So, these are folks that went on and had resolved infection. The tricky thing is, remember I was telling you that just having core antibody doesn't necessarily mean they're immune... they have immune protection, right? So, in immunocompetent

people, those people, if you actually gave them a booster dose or they came in contact with the virus, they're going to mount an anamnestic response, which is characterized by having... You're essentially revving up your B cells and T cells and developing surface antibody all over again. So, you can bring that surface antibody up, which suggests that they're clearly marshaling their memory protection.

And for example, in the liver clinic, when I have patients with hepatitis C, this is another category of patients who often have isolated core. We don't necessarily immunize these folks because we consider them.... Okay, even though they had waning of surface antibody, we consider them immune. Most people out there with isolated core, they're going to fall into that category. You've got isolated core; we assume you have natural immunity. You're done.

Dr. Wood

So, if we're in a primary care clinic caring for people who do not have HIV, who as far as we know, have never had any reason to suspect their immune system has been depleted in any way, and we see an isolated hep B core antibody, basically treat it as immunity because we're not finding surface antibody, but we expect that if they were exposed to hep B again, they'd have this big protective response.

Dr. Kim

Correct. There are two components to immunity, right? We don't want them to get the virus again. We don't want them to develop clinical hepatitis B, right? And so, these folks we don't think are going to develop that because they've got some immune protection. But the other aspect of it is anyone with core antibody has seen the virus before and the aspect of core antibody is hep B does not go away, right. We don't have sterilizing immunity to hepatitis B once we've encountered the virus. The virus has a way of stitching itself into our hepatocyte DNA, both as integrated hep B as well as the cccDNA, which is the covalently closed circular DNA.

So, there's like a stable DNA template that can be elaborated upon and replicated again, so this issue of HBV reactivation is still very much a possibility for people who have isolated core. Like, for example, someone who's in primary care clinic doesn't have HIV, but they later develop some kind of autoimmune disorder that requires them to be on biologics. Let's say they develop multiple sclerosis and needs to be on an anti-CD20 like rituximab or related drugs. These category of biologics have a way of loosening our immune control over hepatitis B and you can develop hep B reactivation, and it's a pretty significant medical event if that happens. It has the potential to cause fulminant liver injury, and in some cases, death if it's not recognized in time. So, someone who is in primary care clinic has an isolated core, you should note that. It should be part of the problem list, hey, if they have an isolated core, there's a potential for HBV reactivation if they're immunosuppressed (with certain drugs).

[considering-hepatitis-b-vaccination](#)**[11:27] Considering Hepatitis B Vaccination**

Dr. Wood

You mentioned for people without HIV, we tend to treat that scenario as likely protected except in scenarios with really significant immunosuppression. How about for people with HIV? How do you think through that and the potential or theoretical benefits of hepatitis B vaccination?

Dr. Kim

Some experts do suggest, and in fact our hep B OI guidelines currently suggest, although the quality of the evidence is not that robust, suggest that these individuals, that hep B vaccination should be considered. Because if you have HIV and have an isolated core, it's not entirely clear that we can assume that same protective immunity. And the idea is, oh, can we use the vaccine to boost that protective immunity? And there have been a few studies that have shown that, hey, when we actually give a dose of vaccine either as a

single dose or as a full series, we can bring that surface antibody level up and therefore potentially improve someone's immunity.

I have some skepticism around that theory for a couple of reasons. One is we don't really have any clinical data to show that when we vaccinate these folks who have an isolated core who've seen the virus before, we can effectively improve their protective immunity such that they don't have clinical hepatitis, or they don't go on to chronic hep B. We just don't have that clinical outcome data. And we also don't have data to say, "Hey, when we give a dose of vaccine, it looks like we prevent HBV reactivation." We don't really have that data. Also, probably because HBV reactivation is a fairly rare event, but that's important to realize is that we're kind of, like, basing this on the fact that, hey, when we give a dose of vaccine, it looks like we bring the surface antibody up, either as a single series or as a complete series.

Dr. Wood

So, we can get the surface antibody level much higher, which might feel reassuring in some way, but there's no real clinical outcomes data to show that that is really meaningful in the long-term clinically, correct?

Dr. Kim

That's correct.

[vaccines-isolated-core-antibody](#)[13:40] **Vaccines and Isolated Core Antibody**

Dr. Wood

And so, there's some guidance that for every individual with HIV who has this isolated hep B core pattern, we should be considering hepatitis B vaccines, maybe recommending it. I hear your skepticism, though. So, in your practice, what are you doing? Are you recommending a vaccine in every instance of isolated hep B core, or when are you really recommending or prioritizing it?

Dr. Kim

I don't do it in everybody. I'll say that off the bat. I look at the individual and figure out what I'm dealing with. The real risk of concern in someone with core antibody is not so much new infection as much as could they have HBV reactivation? And I think where this scenario comes into a little bit more urgency is when we're taking them off tenofovir-based ART. I say specifically tenofovir-based ART because tenofovir is kind of one of our main robust antivirals against HBV and is part of a lot of our current oral ART regimens. A lot of the non-oral injectable ART regimens that are currently available, about to be available, none of them really contain tenofovir. We don't have a long-acting injectable form of tenofovir yet. So, what we're dealing with is you're withdrawing HBV protection from the standpoint of the antiviral because it's clear that there's some data showing that if people who are taking tenofovir, either as part of PrEP [preexposure prophylaxis] or HIV, do appear to have some protection against getting HBV, but in the setting of someone who is core antibody-positive may also have some protection against HBV reactivation.

So, what happens when you take tenofovir away? Well, if someone had not the greatest immune control over their Hep B—let's say they were in that category of someone who had occult hep B or who previously had a chronic hep B and lost their surface antigen, hadn't yet developed surface antibody. When you withdraw tenofovir, if their immune control is not great, you are leaving them open to possibly the hep B reawakening. And we've seen this in people who have chronic hep B mono-infection that are on tenofovir, and we take it off, or any hep B-active antiviral, entecavir included, we take it off, and quite a number of them we're going to see HBV reactivate. We're going to see that the virus come up first, and then the ALT will generally follow in terms of the climbing up. And so, that's a concern in our population of people with HIV as we're thinking about doing these switches, ART switches.

Dr. Wood

And I think those switches are going to come up more and more in the future. I mean, the most recently approved medications, the injectables, as you mentioned, the options that are coming in the near and more distant future generally do not have TAF [tenofovir alafenamide] or TDF [tenofovir DF]. So, what I'm hearing you say, Nina, is if someone has the isolated hep B core antibody pattern and they are taking a TAF- or TDF-based regimen, and let's say they are taking it as prescribed, taking it every day, there are some data that provides protection against new hepatitis B or hepatitis B reactivation. Sounds like in that scenario you're not really pushing hep B vaccination, correct?

Dr. Kim

That's right. Yeah, I don't, generally.

[boosting-immunity](#)**[17:04] “Boosting Immunity”**

Dr. Wood

No, I think that's very clinically relevant and helpful. But then if you are considering a switch of ART, a switch away from TAF or TDF, let's say to a two-drug regimen, whether it's long-acting injectable cab[otegravir]-rilpivirine or oral dolutegravir-rilpivirine or oral dolutegravir-lamivudine, now we have doravirine-islatravir. None of these regimens have TAF or TDF. So, in that setting, you're considering a switch; an individual has isolated hep B core antibody, then what are you recommending?

Dr. Kim

I think you can consider “boosting their immunity.” I put *boosting their immunity* in air quotes because, as I mentioned, we don't really have data to support that we're truly boosting someone's immunity versus just getting their surface antibody titers up. And some of that... I think it's reasonable to consider that rationale because there is data from the VA [Veterans Affairs]. They looked at all people with core antibody. That included some people who had surface antibody and those who were isolated core. This is all people with HIV who switched off tenofovir, either to an oral regimen like doravirine-dolutegravir, or something that just didn't contain tenofovir, and they looked to see, you know, how often is reactivation happening? It turns out it's not that often. It's not that common. The people who apparently were more likely to reactivate were the ones who previously had surface antigen some point in their life. If you went back and looked at all of their earlier serologies, so previously had some evidence of active infection. They also found that the reactivation rate, if they looked at the people who had core antibody and surface antibody, didn't appear to be maybe as high as those who had isolated core.

So, this lack of low surface antibody titer perhaps appears to be associated with maybe a higher risk of HBV reactivation. But this is me saying, but I'm not sure if the corollary is true, which is that if you give vaccination, bring that surface antibody titer up, then they're going to be less likely to have HBV reactivation. We don't know that. And I bring this up because there've clearly been cases of HBV reactivating even when there's surface antibody present with the core antibody. So, on some level, having a positive surface antibody does not appear to confer complete protection against HBV reactivation.

And I remind people about this because we've hung our hats so much on the surface antibody titer, but that's not the whole story when it comes to the HBV immunity. And we know this for a fact. I mean, there's a study from Switzerland way back where they actually looked at people with isolated core, gave them a dose of hep B vaccine, and just looked at what was happening with their B-cell and T-cell repertoire. And there's all this stuff happening that in the background... So, there's all this cell-mediated immunity that we are not measuring that is required—it's almost a precursor before you develop that surface antibody, which is really like the downstream reflection of protection.

Dr. Wood

It's complicated.

Dr. Kim

Yeah. And that might be impaired in someone regardless of what you do with vaccination. So, this is why I'm kind of like... Yeah.

Dr. Wood

Yeah. And this is why I think this is so helpful to hear, Nina. It's way more complex than see an isolated hep B core antibody, vaccinate, watch the surface antibody rise, and then feel good about everything and stop monitoring.

Dr. Kim

Exactly, exactly.

Dr. Wood

... you know, and feel like you're done. Even in that scenario, there is theoretical risk of reactivation, especially with removal of hep B-active agents. This'll come out as audio only, so no one can see your air quotes. But just to reiterate, Dr. Kim was saying we vaccinate and we quote unquote "boost immunity," but we don't really know clinically how much that helps, and it's not 100% protective. Nina, would you agree with that interpretation?

Dr. Kim

Yes, exactly. And that's the other teaching point I would make is that we don't have a full window into someone's hep B immunity. We've seen this just with immunizing people with the standard recombinant vaccine, right? Their CD4 counts have really come up. They look like they're doing really well in terms of their, like, immune reconstitution. And we give them a hep B vaccine, and it doesn't do anything. I mean, we just see this in people who are HBV susceptible that we're trying to immunize, right? And so, this is just me making the point that there's going to be variability and just to have an ongoing awareness around hep B reactivation.

There was a really great [case series](#) that I would point people to worth reading in CID [*Clinical Infectious Diseases*] that got published, I think, earlier this year or late last year by Olcott and others at the University College London. And they go through four cases of HBV reactivation in the setting of ART switches. And it was a very illuminating paper for me on a number of levels. One, because it pointed out, hey, having a surface antibody does not appear to confer a complete protection, hey. And that it was really important to look for recorded history of surface antigen. But they also bring up this idea that we don't really have a good handle or understanding of people's HBV immunity fully.

Dr. Wood

Coming back to the VA data, Nina, it seems like, at least in my mind, if someone has ever had a positive surface antigen, I'd be very hesitant to switch off TAF or TDF, unless I absolutely had to. And even in that setting, you know, I'd really consider monitoring very, very closely or adding other hep B therapy. Would you agree with that if someone has ever had a surface antigen? Positive? Yeah.

Dr. Kim

I think so.

Dr. Wood

Yeah. And then to the point you made, if someone has a positive core antibody and surface antibody, negative surface antigen, even then switching off TAF or TDF, there is still a low but non-zero risk of reactivation. And if someone has a positive core antibody, negative surface antibody, negative surface antigen, they switch off. Again, low, but non-zero risk of hep B reactivation, correct?

Dr. Kim

Correct. Yeah.

[switching-off-tenofovir-what-now](#)**[23:23] Switching off Tenofovir: What now?**

Dr. Wood

So, let's take negative surface antigen, positive core antibody with or without positive surface antibody, and a person really needs to, for whatever reason, switch off TAF or TDF. What is your strategy there in terms of if you're going to offer hep B vaccine, which one, and what does the monitoring for you look like after the ART switch?

Dr. Kim

There are a couple of different ways to approach this. I do think that we need to keep a pretty tight eye on chemistry panel, which I think that people are doing as part of their protocol, for example, with *Cabenuva*. So, it's built into many clinic protocols with *Cabenuva*, so that's nice. Do you need to check a surface antigen or HBV DNA level with every time you check chemistries? I don't think so. I mean, that gets kind of expensive, and I would check both. This is me making a distinction. So, in our clinic, there's the option of doing hep B surface antigen with reflex HBV DNA, right? So, the HBV DNA won't be done unless the surface antigen's positive. I think that's fine, but it's also worth bearing in mind, and the Olcott paper goes over this, is that sometimes you can get an escape mutation where you're not going to get... you're going to get HBV reactivation, so the HBV DNA will be positive, but the surface antigen will be negative or not detectable.

So, if you see someone who has an elevated ALT and you're like, "Uh-oh," and they have a core antibody-positive, I wouldn't just stop at checking a hep B surface antigen with reflex DNA, you know, like I would think about sending both almost kind of separately if you're really worried about it. But in terms of monitoring, like if you're doing that all the time, I mean it's just, it gets a little bit expensive for a fairly rare event. But I do think it's important to keep an eye on the chemistry panel with the LFTs [liver function tests]. And I emphasize this in people that you just know you're taking off tenofovir without the security of the whole *Cabenuva*-like protocol, right? Like, let's say you're switching them to dolutegravir and rilpivirine or dolutegravir-doravirine if they happen to have drug resistance and can't get on cab-ril [cabotegravir-rilpivirine], I mean, like, just keep that in mind. You have to create your own protocol around that.

Dr. Wood

And maybe note it in the chart so everyone in the clinic knows we need to watch out. And any spike in ALT, for example, should make us as clinicians think about hep B reactivation.

Dr. Kim

That's right. And we're used to seeing these folks. I think anyone who gets switched should be (kind of) monitored quarterly for a year. I'm going to maybe press this point a little bit, because we're used to seeing people every six months right now in the HIV world, and we let them go for months without any kind of lab

monitoring. Not sure I would do that in this setting.

Dr. Wood

Nina, let me just... If I can, say that back to you, and make sure I've got it clear and it's clear for listeners too. So, if someone has a positive core antibody with or without surface antibody and they're switching their ART off TAF or TDF, switching to a tenofovir-sparing regimen, consider giving a hep B vaccine to boost their surface antibody up; over 100 seems to be the target, but you're not done. That does not absolutely confer protection. We have a lack of clinical outcomes data. So, monitor, check the AST and ALT, say, quarterly for a year and then go back to every six months, and if there's any spike in ALT, check a surface antigen and consider DNA as well for the reasons you mentioned.

Dr. Kim

I might even consider 18 months until we learn more. I mean, what was interesting about the Olcott paper for me was that a lot of these folks didn't reactivate until the end of that year.

Dr. Wood

Ah, I see. I see.

Dr. Kim

They just happened to get caught with their ALT in the, like, 1000 range or ALT in the 80s. I should mention that, you know, a lot of times HBV reactivation is subclinical. Patients will not come to you with symptoms, and their ALT peaks at like 100 or 50. It can be subtle.

Dr. Wood

I have also seen clinicians monitor the AST and ALT regularly and then just do a yearly check of the surface antigen or DNA; just, no matter what, every 12 months, check that. Do you think that is indicated?

Dr. Kim

I think that's fine. There's a lot of different ways to slice this pie, but I think in that instance you're trying to maybe catch reactivation happening without any ALT elevation. I will tell you the most HBV reactivation happens with ALT elevation. It's really unusual to catch it without. So yeah, sure. If you want to be complete about it, you can certainly do that.

[take-home-messages-closing](#)**[28:21] Take-Home Messages and Closing**

Dr. Wood

I think this is great. Again, so clinically relevant and going to become more and more important, I think, as we have more tenofovir-sparing regimens really become the cornerstone of ART. I think that is going to happen in the next few years. Maybe I could just ask you, and now we'll sum up and just the biggest take-home messages, the biggest thing you would like listeners and clinicians to take from this conversation.

Dr. Kim

I think the main thing is just to have a really firm understanding of what the core antibody represents. And if it helps, put it on the problem list so people know, hey, this person has a history of hepatitis B, and you should put in that, like, you know, for those of you who use Epic, you can put the problem list, and you could put some text around that problem just to contextualize it for someone who's not just kind of taking care of

your patient for the first time. If you happen to know that they had previously a surface antigen that they lost, put it in there. If you have prior records, because people come to us, they don't necessarily start their HIV care with us, right? They didn't necessarily have their hep B serologies with us, but maybe you happen to know this because you looked at their earlier records that are buried deep within the PDF in the media file.

Dr. Wood

Mm-hmm. Happens a lot.

Dr. Kim

Like, put that all in there because we need to bring hep B up so that it's apparent so that we have a really good understanding of where the person stands with their Hep B. If they've had a history of non-Hodgkin's lymphoma with their HIV in the past and got rituximab at some point, that's important to know as well because, you know, these folks, they had their loosening of hep B immunity at some point in their lives, and withdrawing tenofovir, their risk might be a little different than the other person with isolated core. So, these are like qualitative, these nuances around hep B immunity. So, this is me wanting people to have an understanding that hep B immunity is more than just the surface antibody.

Dr. Wood

And I think that is just such a key message there. And then Nina, I will just add to what you said. If someone has a positive core and they've switched off TAF or TDF and then they're going to need rituximab, B-cell depleting therapy, or other immunosuppressive therapy, that's an important consideration as well. And another reason to put positive core antibody or history of hep B exposure, whatever it is in the problem list.

Dr. Kim

Mm-hmm. That's right, 100%. What I told you before when we were looking at the primary care clinic in the general population of just putting that core antibody up there, because these patients may have to get immunosuppressed for a variety of different reasons. We have so many biologics out there, and some of them are going to loosen hep B immune control, even in people with surface antibody that's positive along with their core. So, that same principle applies to people in the HIV care space.

Dr. Wood

Such valuable points. I would also like to mention I will link to the papers we've mentioned in the show notes. I'd also like to mention the paper you wrote recently with colleagues called [Don't Drop the B](#), which I thought was incredibly pertinent to clinical practice and such a great summary of the considerations for the different serologic result patterns with tenofovir-sparing ART or PrEP. So, we will link to that as well.

Dr. Kim

Best tables. So, for those of you who like, just tell me what to do. You could just go to the tables.

Dr. Wood

Yes, the tables are great. So, we will link to the papers we've mentioned here. Nina, thank you so much for all of your incredibly valuable insights today.

Dr. Kim

Great. Thanks for having me, Brian.

[credits](#) [32:09] Credits

Transcripts and references for this podcast can be found on our website, the National HIV Curriculum at www.hiv.uw.edu.

The Health Resources and Services Administration, Department of Health and Human Services, provided financial support for this podcast. The award provided 100% of total costs and totaled \$1,175,136. The contents are those of the author. They may not reflect the policies of HRSA, HHS, or the U.S. Government.

[core-antibody-persons-hiv](#)