**Implementing the AADE Practice Advisory for Diabetic Kidney Disease**

**Andrew S. Narva, MD, FACP, FASN**

Thanks a lot. I'm really glad to have this opportunity. I have a lot of stuff I want to talk about. So I'm going to be very brief. I worked for Indian Health Service for 25 years and about 10 years ago, got asked to come to the NIH to run the National Kidney Disease Education Program. I've continued to serve as the Chief Clinical Consultant for Nephrology for Indian Health Service and I continue to see patients at Zuni by telemedicine, which has been very nice. I'm just looking at the list of attendees and I see a lot of familiar names which is very nice.

I'm going to talk about diabetic kidney disease. I'm going to talk somewhat about the AADE Practice Advisory because we helped write that and we've developed some tools to help implement that.

Briefly, I will talk about the burden of kidney disease due to diabetes in the U.S., how we define diabetic kidney disease and how we assess the risk or progression. Interventions which we know can slow progression of diabetic kidney disease. Very briefly, I mentioned the AADE Advisory and I want to describe a curriculum that we've developed for diabetes educators that's available for free to all of you.

Before I proceed, I serve on the Nephrology Subspecialty Board of the American Board of Internal Medicine and I have to show this slide in case anybody is listening and hopes to learn questions or answers to the next certifying exam. You will not find it in this talk, but I don't think that's a big issue.

So what is chronic kidney disease? Prior to about 15 years ago, there was really no shared understanding of what chronic kidney disease was. So people had all kinds of terms, renal insufficiency, mild renal failure, and no one had a clear idea of what we were -- there was no shared understanding what we were talking about. As a result, there's a real barrier to making anything better in implementing standards of care or strategies for improving outcomes.

About 20 years ago, there is a consensus that CKD could be defined by two different criteria. The first is a functional criteria and the kidney functions as a filter. So, based on a decreased filtration rate, decreased on the amount of blood that's filtered, you can diagnose someone who's having chronic kidney disease. It's generally about 60 milliliters per minute, which is about half normal.

The other way of identifying people with chronic kidney disease is based on kidney damage. And kidney damage can be anything. It can be abnormal x-ray in the past, biopsy 20 years ago, proteinuria, and hematuria. But for the most part when people talk about kidney damage, they're talking about proteinuria. Proteinuria is generally measured as albumin, which is the main protein in the urine. If someone has more than 30 milligrams of albumin in their urine for 24 hours, they will be diagnosed as having chronic kidney disease.

What does this all mean? Chronic kidney disease in general means you have fewer functioning nephrons and don't worry, I'm not go through all this anatomy of the nephron. But just to remind you that each -- the kidney is a filter, when we talk to patients and try to explain what's going on, it's important to convey the idea that the kidney is not one large filter. Each kidney is made up of about a million filtering units and they tend to be destroyed one by one.

Because of the way that we're built, there's a large, what we call physiologic reserve. And what that means is, we have a lot more filtering capacity than we need to survive. What that means is you can lose a lot of kidney function. You can have a lot of kidney damage without any symptoms. And most people don't really have any symptoms until they've lost more than three quarters of their kidney function.

Why is that important to all of us who are trying to explain kidney disease to people with diabetes? It means that if we want to intervene early, we're almost always talking to someone who has no symptoms. And because kidney disease can be frightening, you're often in the position of telling someone information that is frightening at a time when they don't have any symptoms. They may not like that and they may get angry about that.

How do we assess kidney function? We're going to talk about assessing kidney function and assessing kidney damage. Kidney function is generally expressed as the rate at which blood is filtered. We call it the glomerular filtration rate. The GFR is the sum of the filtration rates of all two million functioning nephrons. Each one filters a tiny amount. All together, they filter quite a bit. We can't really measure this. We do estimate it from laboratory tests. And the estimated GFR that you see on all laboratory results within Indian Health is an estimate. And that's just important to remember. If not the measured GFR, which is a tricky thing to do and somewhat complicated, and I'll explain why this is important in a minute.

How do you get an intuitive sense of what a normal GFR is? Well, if you think of the heart as pumping about 6 liters of blood each minute, about 20% of that cardiac output, which is about 1.2 liters per minute goes to the kidneys. So the kidneys get a lot of blood flow. What gets filtered, of course, is not the blood cells, what gets filtered is the plasma. And plasma makes up about half of blood volume. So that's about 600 milliliters per minute of plasma is flowing to the kidneys. Now, what the kidneys filter, they can't filter all of that plasma because that would leave 100% of blood cells in the capillaries in the kidney and 100% blood cells are not going to flow. They won't be able to move. So, only about 20% of the plasma gets filtered and 20% of 600 is 120 milliliters per minute. And that's roughly what a normal GFR is, 120 milliliters per minute. So that's quite a bit.

How do we get a sense of what the GFR is? Well, in the past, we just looked at the creatinine and the creatinine is a waste product from muscle. And the creatinine is excreted by the kidneys. The creatinine will go up as the filtration decreases. In the past, we often just looked at the creatinine and made some kind of assumption about what the kidney function was. The problem is because creatinine is a by-product of muscle metabolism, if you have more muscles, you'll have a higher baseline creatinine.

For example, a body builder who is 6'6" and 260 pounds of pure muscle will have a higher creatinine than a small, non-muscular person who might be 5'2". They both might have the same level of kidney function but their creatinines will be very different. Because the creatinine by itself can be misleading, we found other ways to make a better estimate. Using just the creatinine though, there's an example at the bottom of the slide that shows if you have a 28-year-old African American man with creatinine 1.2, his estimated GFR is 60. If it's a 78-year-old white woman with a creatinine of 1.2, it's 43. Those are very different figures. Looking at the creatinine by itself is maybe misleading. And in fact, if you're still confused a little about the estimated GFR, it's greatest utility is in understanding that what looks like a "normal creatinine" may actually reflect significant kidney damage.

So, we use an estimate in the equation to estimate the GFR. That's the small eGFR, is the estimated equation. And one of the equations is this Modification of Diet in Renal Disease, the MDRD. Most IHS labs are in the process of switching to another one. It's not really important to look at the equations. In fact, looking at them will just remind you how much math you forgotten since high school.

The important thing is that both of these equations have four variables. They have the serum creatinine, they have the age, they have the gender, and the race. Gender, race, and age are three factors which affect muscle mass. So what the equations do is they correct for the variation in muscle mass between individuals. People who are younger have more muscle mass than people who are older. People who are male tend to have more muscle mass than females, and African Americans have a slightly higher percent of their body as muscle mass than Caucasians and other races as well.

The thing is the eGFR estimates, the measured GFR. The measured GFR is the actual GFR. These equations are -- they're derived from population-based studies. So in other words, they do these equations -- they develop these equations in research populations where people have had a measured GFR. They look at demographic data on those patients into something called regression. They developed an equation which gives the best estimate of -- the best match for the measured GFR. And that's great, it's very helpful. But it's important for you and when you're counseling patients, to understand that although the estimated GFR is better than just looking at the creatinine, it's still kind of an imprecise measure. The standard that we assess these estimated equations is called the P30. And the P30 is the percent of GFR estimates that are within 30% of the measured GFR or the actual GFR.

You may find all this quite confusing. So what does it mean? It means if you get a result for a patient and the estimated GFR is 59, it means there's about an 80% chance that the measured GFR is between 42 and 78. It's giving you -- it's the best guess we have but it's still just an estimate. And you want to be real careful because above 60 is actually normal and you want to be very careful about diagnosing somebody as having chronic kidney disease, if all they have is one estimated GFR that is just a little bit below normal. Because even though it's not as precise as we'd like it. It's the best thing we got but it's still not precise.

The estimated GFR, once again is not the actual or measured GFR. It's a very good estimate of the risk of actually having decreased kidney function. But like other risk predictors, you want to be very careful in diagnosing someone with a disease if that's all you have. If you happen to see people who are not diabetic and they have no proteinuria, and they have no high blood pressure, and they have a GFR of 55 -- estimated GFR 55, you might want to be real careful about telling them they have chronic kidney disease until you get more additional information or you repeated the study.

The other issue that we have is that these estimated equations are much less reliable at more normal GFRs and also, the kidney function tends to decline with age even though the diagnostic threshold is the same. Again, we want to be really careful. If you see someone who's 78 and their GFR is 55, and they don't have anything else to suggest kidney disease, I think there's a lot of controversy about whether that person has chronic kidney disease or has anything that's really likely to compromise their health.

So why am I spending so much time on this, it's because increasingly, we have performance measures and quality indicators that are based on lab results. Unfortunately, we sometimes do things to people based on a single lab result on the basis to reach a performance measure and it may not always be the best for the patient. Like everything else that you look at with patients, you have to look at the whole picture not just one measurement.

In addition, any estimate of kidney function based on creatinine has to be used with caution in certain situations such as, any time the creatinine level is changing you will not get an accurate indication of kidney function. And that includes many people who are sick enough to be in the hospital. Also, people who have really extremes in muscle mass or body size, someone who has two amputated legs or on the other hand, it's just a huge individual, you need to be really cautious about interpreting their creatinine or any estimate based on that. And also, some medications will interfere with the measurement of creatinine.

How do we explain all this to the patients? What we do is we say, above 60 here, on this speedometer is normal. Now actually, what that means is 70 in an 18-year-old is not normal but the estimates up here are so variable that they can't be distinguished from normal. But we just say if it's above 60, it's normal. If it's between 15 and 60, we say the patient may have chronic kidney disease based on GFR. And if it's less than 15, there's sort of an arbitrary decision to describe that as kidney failure.

What about kidney damage which is the other way that we assess people for the presence of chronic kidney disease. We basically use urine albumin to screen and identify, to diagnose and to treat diabetic kidney disease. And as I'll show you later, an abnormal urine albumin level is often the earliest marker for kidney disease complicating diabetes. It often shows up before any noticeable change in creatinine or GFR. Beyond that, it is a very important prognostic marker especially in diabetes.

So someone who has diabetic kidney disease, retinopathy, and high levels of protein, albuminuria, is in real trouble, almost regardless of what their creatinine is today. It can be used to monitor and guide therapy. And finally, it's a really excellent tool for patient education and self-management. And informing the patient of the level of albumin in their urine is no different than trying to explain hemoglobin A1C, estimated average glucose or estimated GFR. If you can say to a patient, "Before we started you on the Lisinopril, and got your blood pressure under control, you had half a teaspoon of albumin in your urine everyday and now it's a quarter. And we think that that really makes a difference and that's a sign that you're doing better."

We measure, we assess the amount -- we estimate the amount of albumin excretion in 24 hours from a spot urine albumin to creatinine ratio. This is something that we all do, but many people do not understand what it means. This uses spot urine specimen and it's useful because the ratio of albumin to creatinine in a spot specimen is roughly the same as it is in 24 hours. So if you have twice as much albumin as creatinine in the spot specimen, you will have twice as much in 24 hours. Why is that helpful? It's helpful because in 24 hours, you have about a gram of creatinine. As a result, if you get a ratio and it's usually reported as milligrams per gram, milligrams albumin per gram creatinine, which is equivalent to milligrams of albumin per day. If you look at the report on your lab report and it says, 722 milligrams per gram, that means 720 milligrams -- whatever it was, 720 milligrams of albumin per day. That's all it means. It's an estimate of 24-hour excretion based on what you see in the spot specimen.

Now, urine albumin is a continuous risk factor and what that means is some of the terminology that we've historically used is probably going to go away, hopefully will go away, because it's very confusing to many clinicians. The term microalbuminuria is it has been used to describe 30, which is the upper limit of normal, to 300 and then greater than 300 is macroalbuminuria.

Now, the 300 cutoff is really just the function of what the dipstick used to pick up. The urine dipstick picks up about 30 milligrams per deciliter or 300 milligrams per liter or approximately, 300 milligrams per day. So that cutoff just is a historical remnant of when we just had dipsticks. There is nothing magical about when you go from 280 milligrams today to 320. It's a continuous thing, 400 is worse than 300, 300 is worse than 200. And probably, what we will come to, is just the urine albumin and if it's normal, it will be below 30. If it's above that, it's abnormal.

How do we explain urine albumin to patients? We spent a long time developing this little graphic and this is an attempt to explain it at a level that most people can understand. This shows a normal kidney. This shows kidney filter, and this shows the albumin passing through and into the urine. And these figures are from patient handouts that we've developed and I think are used in IHS as well.

Based on these two criteria, an eGFR less than 60 or urine albumin greater than 30 milligrams per gram, 10% of U.S. adults may have chronic kidney disease. There are folks or advocacy organizations that would like to identify as many people as possible in the universe as having chronic kidney disease. But it's probably somewhat less than this. You notice here, a lot of these patients are elderly people.

And they may just have decreased kidney function associated with age. But clearly, it is a serious problem and it's a growing problem and it's a problem that can be addressed.

Now, diabetes is the leading cause of kidney failure with end stage ESRD is end stage renal disease or a kidney failure requiring dialysis or transplant for treatment. This curve, it shows that in the last couple of decades, all of the growth that we've seen in ESRD has been due to diabetic kidney disease. And this is mostly type 2 diabetes.

What's the natural history of kidney disease and diabetes? This shows the years of high blood sugar, starting at the onset of diabetes. This shows GFR and this shows urine albumin. So initially what happens after you develop diabetes is your GFR actually goes up. It's a part of the physiologic response to having a high sugar and your kidneys tend to reabsorb the sugar along with sodium and water. And so you get a little volume expanded and your GFR goes up. Then after about 10 to 12 years, the GFR starts to decline. And right around the time it reaches where it started off, still looking quite normal, the urine albumin starts to go up. So the increased urine albumin, the microalbumin as people used to say, is often the first sign. Because people say the GFR is normal, it is at the normal level but it's actually come down significantly from up here. And then over the next 10 or 15 years, the GFR comes down in a pretty steady way. The urine albumin goes up and then comes down when the GFR gets so low that not much urine albumin is filtered.

What's the prevalence of diabetes in the U.S.? It's not just American Indian people and Alaska Natives. There are, as you know, approximately 30 million people with type 2 diabetes. What percentage have kidney disease? About 35%, 17% have just albuminuria, another 11% have decreased GFR, another 7% have both. Well, most of these people are not going to go on to develop kidney failure requiring dialysis.

So why is kidney disease so important? Well the reason is that kidney disease is very much associated with the increased mortality in type 2 diabetes and mostly cardiovascular. So if you look at the 10-year mortality, this is the baseline in the general population. People with diabetes and no kidney disease, the risk isn't hardly higher than it is among people without diabetes. They have albuminuria, it increases significantly. If you have decreased GFR, it increases significantly. And if you have both, it's way up. So kidney disease is a marker for cardiovascular disease and in fact, most people with progressive kidney disease will die from a cardiovascular event and they have a much greater chance of that than going on to dialysis. But many people are going on to dialysis and you can see that during the past few decades, the number of people on dialysis is gone from about 100,000 to about 400,000. That's a lot of people, that's a lot of suffering.

Why is it so important for all of you to understand what you can do about a diabetic kidney disease? Diabetes is the most common cause. The rates have been going up and the reason is, is that by intervening appropriately, you can reduce the rate at which people reach end stage renal disease or kidney failure. And in fact, many people may never reach that point.

In addition to avoiding suffering and being on dialysis, there's a significant benefit to the healthcare system. It costs about $80,000 to take care of someone each year on dialysis. To take care of someone who has advanced chronic kidney disease but is not on dialysis is about $20,000. So for every patient, every year you keep a person off dialysis, you've save the healthcare system between 40 and 50 grand. That's useful because that money can then be reprogrammed to prevent the next generation from getting diabetes in the first place which is of course, the answer to all of this.

What are the key issues in managing diabetic kidney disease? You want to ensure that the diagnosis is correct. Just because someone has diabetes and kidney disease doesn't mean their kidney disease is from diabetes. People have lupus. They can have multiple myeloma. They can have all kinds of things. You need to monitor progression. You need to implement appropriate therapy to slow progression. You want to avoid the acute kidney injury, which means not -- I'll explain it, what that's

about. You need to screen for complications, educate the patient about CKD and prepare them appropriately for kidney failure. And these two go together because kidney disease is complicated and the treatment is scary. For people who are going to go on to dialysis, it's really critical that they get information and have time to understand it so they can make their own choices about how they want to be treated. That process needs to start well before the patient gets referred to a nephrologist.

For those of you listening who are diabetes educators, you are trusted sources of information. And you may be able to communicate complicated and scary stuff to patients in a way that the busy nephrology consultant may have trouble doing. So you can also do it at a point where the patient really has time to understand, to ask questions, think about it, and come back in three months. That makes a huge difference.

What can we do to slow progression? We can treat hypertension. We can treat diabetes especially early in the course. We can lower urine albumin and we can treat the cardiovascular risk factors, because that's what people with diabetes and kidney disease die from.

Blood pressure in people with kidney disease is not good. A significant proportion of people have blood pressures that are not well-controlled and it gets worse as GFR declines. The target right now is 140/90 based on JNC 8. There are obviously discussion about lowering and changing it but as you know, the issue for many people is the patient with a blood pressure of 160/110 and getting that down. There's no controversy at all about doing that. There may be different standards in the future for people who have high levels of proteinuria. You may have heard about the SPRINT trial which came out recently which recommended a blood pressure goal much lower of 120 systolic but that study included no diabetics.

So how do you do this? The DASH diet which many of you probably are much more familiar with than I am in terms of actual implementation is a useful diet and it's a helpful diet. It's been shown to work. It's not widely used in CKD because it tends to involve a little bit higher protein intake, higher phosphorus and higher potassium. Those are not necessarily problems for people with earlier kidney disease and it's a diet, though, that you can implement. And a diet you can implement is better than a more ideal diet that you don't implement because it's too hard to follow.

What about glucose control? There's evidence that control of newly diagnosed diabetes may help prevent CKD, both in Type 1 and Type 2 diabetes. However, once kidney disease is established, tight control, the evidence that tight control has benefits over average control, is much less. There is a problem. Hyperglycemia does harm the kidneys; however, as kidney disease advances and the intensity of glycemic control increases, there's an increasing risk of hypoglycemia. All the evidence we have that shows an effect is based really on the progression of proteinuria. We have not really been successful in showing compelling data that tight glucose control reduces the number of people who will actually reach dialysis. So we have to balance the risk of intensive glycemic control against potential harm, and I'm sure you're all familiar with individualizing goals.

The other issue that you should be aware of is that since insulin is metabolized by the kidney, insulin lasts longer in people with more advanced kidney disease. And if you're seeing someone who's had no change in medications, no change in weight, no change in physical activity, has some kidney disease, and glycemic control has gotten better, you should really think about whether their kidney function has gotten worse.

So we individualize the A1C goal in patients and I know this is a standard in IHS as well. The goal for the general population is generally less than 7, but you want to be much less stringent in patients who are high risk for hypoglycemia, have limited life expectancy and if they have multiple complications, you may be causing more harm than good by being overly aggressive.

In terms of weight loss, high protein diets to improve glucose control have a potential risk for kidney disease because high protein raises pressures and flows in the kidney that may cause damage in a similar way to diabetes. What I tell patients is that after one year, there's no difference between just a balanced but lower calorie diet versus a high protein diet, and since there's a potential risk of a high protein diet, it's better and probably more consistent with life as we know it to follow a healthy balanced but hypocaloric diet if they want to lose weight.

Now, what about albuminuria? You can see here that the risk of progression of kidney disease goes up as albuminuria goes up and as GFR goes down. And the risks are highest in people with both decreased GFR and decreased albumin. A renal event is loss of half of kidney function, dialysis or death. This schematic shows why urine albumin is important to measure and to keep measuring. This shows the risk of a renal event and the albumin at the diagnosis, in a study that people were followed for quite a long time. And so it shows that as the albumin at the time of diagnosis goes up, the risk of a renal event also goes up. This is another study that looked at the response in decrease in albuminuria following the starting Losartan. So as you go in this direction, you have a bigger response to Losartan. This again has the risk of a renal event. What this shows is the greater the decrease in urine albumin, the less likely someone was going to go on to something bad.

So it's a standard in IHS, in everywhere actually, to measure urine albumin at one point. There is push-back; people say, "Well, look, I measured urine albumin and it's elevated. I started them on an ACE inhibitor. Why should I ever measure it again?" It's useful because if you see this kind of response, you can feedback to the patient and say, "Look, your urine albumin is going down. Everything we know about how kidney disease works suggests that your kidneys are benefiting." And I think that's a useful tool. It also gives you an idea of how to manage people. I can tell you that patients that I have put on maximal therapy who continue to have high levels of albuminuria, I use that as an indicator to prepare them for kidney failure at an earlier time than for people who have lower levels of albumin, or have responded to treatment.

So, what can you do to reduce urine albumin? Reduce blood pressure, reduce salt intake, good control of diabetes early, weight loss, excessive protein intake reduction, and stopping smoking. As you all know, probably angiotensin-converting enzyme inhibitors, lisinopril and others, as angiotensin-receptor blockers like Losartan lower blood pressure but they have a special effect on the kidneys that are protective. And so they will reduce the protein in the urine and they are so effective that they are sometimes prescribed to people who have high levels of albuminuria even without blood pressure elevation.

Managing cardiovascular risk, this shows the effect of urine albumin and decreased GFR on cardiovascular risk. These are events, same thing, as urine albumin goes up, whereas GFR goes down, the risk of a cardiovascular event goes dramatically up, and again this is what most people with kidney disease die from. What can you do? Well, lipid abnormalities tend to increase as GFR goes down and so what we do do is we treat with statins for people with CKD. Statins have been shown to reduce cardiovascular risk in people with CKD, although they do not slow the progression of kidney disease.

Now, what are the complications of chronic kidney disease? I'm just going to mention these because the treatment for many of these is still controversial. Anemia because erythropoietin is produced at a lower rate. Hyperkalemia which you see commonly in people with diabetes and on Lisinopril or Losartan. Malnutrition, metabolic acidosis which is serum bicarb less than 22 and this can often be treated with sodium bicarbonate which is cheap and easy. Metabolic bone disease is quite common. The treatment is complicated and the evidence for the effect of this treatment is really lacking. There are many drugs, very expensive drugs that are advocated to use in that situation, though I'm not going to go into those.

Now, finally, one intervention that is very important is acute kidney injury. Acute kidney injury is the sudden loss of kidney function, not necessarily a huge amount, either increase in creatinine of greater than 0.3 milliliters/deciliter, or 50%, and it's significant contribution to admissions from the outpatient center. Drug-induced AKI accounts for about 20% of hospital admissions for AKI. And it's much increased in the elderly population similar to what many of you see.

So who's at risk for acute kidney injury? People with diabetes and hypertension, people with multiple comorbid conditions including congestive heart failure, liver disease, people who were recently discharged from the hospital with multiple comorbid conditions and people who require the use of drugs that affect renal hemodynamics; and those drugs are ACE inhibitors, ARBs, diuretics, and NSAIDs. All of these are used widely in the population that's served by Indian Health.

There is a video which I'm not going to show because I don't really have enough time. It's on our website. It's two and a half minutes and it really goes over how ACEs and ARBs, and NSAIDs, and volume depletion interact to cause acute kidney injury. I think you'll find it useful and I think Jan may get you the exact link. If not, it's on the NKDEP website.

Kidney failure is a GFR less than 15. It means kidneys can't maintain homeostasis. And it's associated as a result with electrolyte and hormonal imbalances and metabolic abnormalities. End-stage renal disease, which is an administrative term, it means the patient's on dialysis or has a kidney transplant. So you can have kidney failure and not have end-stage renal disease. But of course, everyone with end-stage renal disease has kidney failure so they're not the same. They shouldn't be used interchangeably.

You may or may not be aware that kidney disease education is a Medicare benefit for people on Medicare with a GFR less than 30. Unfortunately, the way this was written, the eligible clinicians are physicians, PAs, nurse practitioners, and nurse specialists. It does not include dietitians. It does not include pharmacists and that may be one of the reasons why this benefit is hardly used at all.

What are our goals for population management for diabetic kidney disease? First is to recognize and test at risk patients. That really means a yearly eGFR and a yearly UACR. Screen for complications, which really just means the routine biochemical screen plus a phosphorus, a hemoglobin, and maybe a PTH. Treat cardiovascular disease which means treating hypercholesterolemia and getting people to stop smoking. Referring a patient to a dietitian because dietary interventions are very effective in diabetic kidney disease. Avoiding acute injury and this plays a significant role in advancing kidney disease. All of these things are basically routine diabetic care. There's nothing about this that you aren't already doing. The most radical thing you could do is actually to educate the patients about CKD and its treatment. Because in many cases, telling people they have CKD is akin to telling them they have cancer and if you have a limited amount of time to see a patient, it's not something you can mention in the last minute. It requires explanation. Oftentimes, it doesn't get explained at all.

Just to give you an idea of the need for better education, this shows that only about 20% of people who've lost half their kidney function are aware that they have kidney disease. Which means they're answering yes to this question: Have you ever been told you have weak or failing kidneys?

Well, the answer from the kidney community is early referral. You can see here that still, even people who've seen a nephrologist for over a year, a significant proportion of them had no understanding of treatment modalities and at least a third didn't even understand their diagnosis. These are people who have been referred to a nephrologist. That's one study with nearly 700 patients.

Another study with 400 patients showed basically the same thing, a third weren't aware of their diagnosis, a third didn't understand that it could affect their heart disease risk, they didn't understand how the kidneys worked, the third didn't even understand terminology. So, sending patients to a nephrologist is important when it's appropriate but it's not going to solve the problem. One of the more

shocking pieces of information is the low percentage of people who have seen a dietitian. Only four percent of people starting dialysis had ever seen a dietitian more than a year before they started dialysis. Only about 13% of people starting dialysis had ever seen a dietitian and this is a kind of treatment that's very effective.

Historically, kidney complications of diabetes have been the complications that people sort of stayed away from. Even when people are putting a lot of effort into screening for neuropathy and screening for all the other complications, the kidney disease complications were somewhat just not addressed as intensely. The diabetes community is being more aggressive now and including diabetes educators and we've collaborated with them and others to help develop this practice advisory which came out a few years ago and actually has gone through a revision.

There are four basic tenants which look somewhat similar to what I've mentioned earlier. The first is to identify CKD due to diabetes and educate the patients about their test results which means you explain estimated GFR, you explain UACR. Slow the progression of diabetic kidney disease through the blood pressure control, glucose and control of diet. Collaborate with primary care clinicians to identify and monitor complications, anemia, malnutrition, bone disease. And then Promote self-management. Talk to patients about CKD, communicate the importance of what testing means and the results, explain that it's a progressive process. You can't make the creatinine go back to normal the way you can make glucose go back to normal. GFR will continue to decline. The goal is to slow progression.

And begin speaking about dialysis and transplantation. Many of you may see this as not part of your job but in most cases, you are the trusted, consistent provider and who better for a patient to hear about something that's scary and complicated than from you? You really don't want to wait until the patient's referred to someone they don't know who's maybe very short on time and may not be so oriented towards explaining things in a way that the patient can understand.

Along with this advisory, we developed a four-module training program for diabetes educators. This is our logo. Oh, there it is. This is our logo up here and the four modules are aligned along the tenants of the advisory: identify diabetic kidney disease; slow progression; complications; and treatment choices for kidney failure. This curriculum is available on our website which now looks like this and here's the link. Each module is about an hour and you can listen to the narration. Some of it is in my voice which you may or may not like. You can do the training. If you want to get credit, continuing education credit from AADE, you can go to their website and do the same curriculum but you'll get credit. In both cases, you have the opportunity to take self-evaluation questions to assess how well you've understood what was discussed.

If you want to go beyond that, we have a short, about 16 pages, booklet for primary care providers on managing adult CKD patients and this is something available on our website. You can order or you can print it. There are lots of educational materials for clinicians that's been produced. Much of it is way too voluminous and is addressed as if patients with kidney disease were the only patients you see. This is written with the understanding that kidney patients will be a fraction of the patients you see, that you have many other things to do, that you need a clear and simple way to address the key needs of these patients.

One of the lessons that I learned in Indian Health Service and that we're trying to address through our program is that CKD is best addressed through population management and better outcomes will occur when it's implemented in the community and in the clinic by everyone in the clinic. It's really going to be most effective if everyone and all the healthcare providers in the clinic, the pharmacists, the dietitians, nurses, nursing assistants, if everybody has an idea what this problem is about, like they do about diabetes, they can address it.

The easiest way to implement systematic change is not to have a kidney clinic. The easiest way and IHS is the model for this, is to modify the diabetes care delivery system so that it does a little bit better

job addressing kidney complications. It's important to remember that the kidney complication shouldn't be addressed by itself but as part of -- as this multi-system disease. And then the emphasis should be on ensuring that the patient receives care from someone who's competent and interested, not focused on referral. Referral is important but if all you do is refer the patients, you may have already missed. If you wait until the referral time, which for many folks is 30 milliliters/minute of GFR, by then the game is pretty much over, and you've missed a lot of opportunities to make a difference. And those differences early on have the biggest impact.

How do you know I'm not just some guy at the NIH spouting fiction from the ivy-covered walls? This chart looks at the rate of new cases of end-stage renal disease by race in the U.S. And you can see that these curves tend to be fairly flat. Prior to 1996, all of them were going up. This one is American Indians and Alaska Natives. What you can see here is this rate, which was even higher before this and was about three times what it was among the white rate, has gradually gone down and is now almost identical to the white rate. So if you're an American Indian, your risk of developing kidney failure is not much greater than it is for Anglos. Now it's gotten worse for Anglos but slightly worse over time, but the rate has come down dramatically and what this reflects -- I can't prove this but what it reflects really is the success of a comprehensive diabetes program in Indian Health, in IHS, in tribal programs, in urban programs. And although it's not perfect, it's a systematic way of addressing diabetes through a team method. I think this curve is all about that. So much of what I do is go around the country and talk about what IHS is and it has accomplished. But there it is. That's hard data. Although some days it may feel like you're not being as successful, if you look at the entire population, it's better and the results are much better.

So it's 3:59, or whatever it is where you are, it's 59 minutes past. I've used up almost all the time. I realize that I don't have time -- there's very little time to answer questions. Please write me if you have a question. I have spent the last 30 years answering questions for IHS clinicians and I learn a lot and it helps me do my job better, and all the materials that I showed you are available in this website. So thank you very much for your attention and continue the good work.