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Evaluation and Management of AKI in Cirrhosis: At the Interface of Gastroenterology and Nephrology

Announcer:
Welcome to CME on ReachMD. This activity, titled “Evaluation and Management of AKI in Cirrhosis: At the Interface of Gastroenterology and Nephrology” is jointly provided by CiME and NKF and is supported by an educational grant from Mallinckrodt Pharmaceuticals. Before starting this activity, please be sure to review the faculty and commercial support disclosure statements as well as the learning objectives. Here’s your host, Dr. Brian McDonough.

Dr. McDonough:
This is CME on ReachMD, and I’m Dr. Brian McDonough. Joining me to share their perspectives on evaluating and treating acute kidney injury, or AKI for short, in cirrhosis patients are doctors Nancy Reau and Kevin Regner.

Dr. Reau is a professor of internal medicine, the Richard B. Capps Chair of Hepatology, Associate Director of Solid Organ Transplantation, and Section Chief of Hepatology at Rush University Medical Center in Chicago.

Dr. Reau, thanks for being here today.

Dr. Reau:
Thank you so much for inviting me.

Dr. McDonough:
And Dr. Regner is a nephrologist at the Medical College of Wisconsin in Milwaukee, Wisconsin.

Dr. Regner, it’s great to have you with us as well.

Dr. Regner:
Happy to be here. Thank you for the invitation.

Dr. McDonough:
So, if we start with some background, Dr. Reau, how can decompensated cirrhosis and increasing portal hypertension impact kidney function?

Dr. Reau:
It’s really important to understand that cirrhosis is a spectrum. So, lots of times when a patient is labeled as a cirrhotic, they get upset and nervous, but that’s a really, really big word, and with asymptomatic cirrhosis or compensated cirrhosis, you can have portal hypertension, but the patient doesn’t have any symptoms of liver disease. So, the liver can be nice and stiff, you can have a gradient across that, which increases potential risk, but the patient may not know that.

Now, if you have a decompensated cirrhotic, so that’s someone who has symptoms of liver disease. They have ascites or fluid in their belly. They have varices that have been symptomatic, they’ve thrown up blood, or they have encephalopathy, then these things all make that patient very fragile. And you can imagine that across that spectrum, as things become more and more progressive, the

pathophysiology that impacts the kidneys is getting worse. And so, that splanchnic vasoconstriction that is making the kidneys not get as much nutrition is really escalating to the point that any little event in that person's environment can really push the kidneys to fail.

Dr. McDonough:
And turning to you, Dr. Regner, can you tell us and assess in how you asses for AKI in patients with cirrhosis?

Dr. Regner:
Sure. So, acute kidney injury is defined by changes in serum creatinine. And so, in recent years, the definition of acute kidney injury in patients with cirrhosis has been now aligned with the definition that we use in the general population. Prior definitions of AKI in patient cirrhosis required the patients to achieve a serum creatinine rise above 1.5 mg/dL. But now we've changed that definition to, again, as I said before, to that which we use in the general population of a change in serum creatinine of a 0.3 mg/dL rise within 48 hours.

And so, that's now allowed us to capture AKI in patients with cirrhosis much earlier in the course of the disease. It also allows us to identify acute kidney injury in patients with cirrhosis who may have low baseline serum creatinine levels because of low muscle mass, poor nutrition, reduced conversion of creatine to creatinine, or larger volume of distribution of creatinine due to ascites and edema. And so, this newer definition of AKI now allows us to identify patients earlier with the hope that we might be able to treat them earlier.

Dr. McDonough:
So, there's a lot of different mechanisms behind the different types of AKI?

Dr. Regner:
Right. So, if you think about the three most common causes of acute kidney injury in patients with cirrhosis, prerenal AKI, acute tubular injury, and hepatorenal syndrome. There's also a multitude of other causes of AKI that we see in the general population. So, if we think about prerenal AKI, that might occur in patients with cirrhosis due to the use of diuretics or lactulose or to blood or fluid losses.

Acute tubular injury can occur due to prolonged ischemia or due to the use of nephrotoxic drugs. HRS, of course, we will talk about, I think, later. And then, of course, there's a bunch of other causes of AKI that we see in the general population. Patients can have obstruction. They can have glomerulonephritis. They can have glomerulonephritis that's due to specific causes within the cirrhotic milieu, like IgA nephropathy or hepatitis C-associated glomerulonephritis. And so, the differential diagnosis of acute kidney injury in these patients is broad and really requires us to perform a comprehensive diagnostic evaluation.

Dr. McDonough:
Coming back to you, Dr. Reau, can you tell us how you approach a differential diagnosis for a patient with AKI in cirrhosis?

Dr. Reau:
Yeah. It's absolutely important, like, with just outlying, there is more than one reason that a person with cirrhosis can end up with a change in their kidney function, and kidney function fluctuations are very common in our population. So, one of the easiest things is to know what the prior kidney function was. So, sometimes you do have patients that come in and they haven't been seen in months, and you don't have a understanding of what their recent baseline is. But if you know what their kidney function was a couple weeks ago or a month ago, then you have a better understanding of what's occurred more recently.

After that, you want to absolutely look at medication changes. Did the person just have big, large-volume paracentesis? Did they get dehydrated because they were out taking their diuretics, and it was a really humid, hot day? And understanding what else might have happened to that patient. Did they recently have a GI bleed? And so, that kind of helps you approach the patient to understand if it's a progression of their liver disease or if it's something else that's secondary that you might want to reverse first.

Dr. McDonough:
So, based on your assessment, it isn't just creatinine. There could be other markers or other things you're looking at.

Dr. Reau:
100%.

Dr. McDonough:
For those just joining us, this is CME on ReachMD. I'm Dr. Brian McDonough, and today, I'm speaking with doctors Nancy Reau and Kevin Regner about the evaluation and treatment of acute kidney injury in cirrhosis.

So, Dr. Regner, if we switch gears and focus on treatment strategies, do you treat different etiologies of AKI?

Dr. Regner:
Right. Going back to what I mentioned before, it's very important to identify the underlying cause of the AKI so you can treat that particular cause appropriately. So, for example, with a obstruction or glomerulonephritis, we would treat that just like we would a patient

in the general population; commonly, what we're left with in patients with cirrhosis and dealing with prerenal AKI, acute tubular injury, and hepatorenal syndrome.

So, with prerenal AKI, we treat the volume depletion with either crystalloids or colloid fluids, including IV albumin, with the hope that that will reverse the kidney dysfunction. There's no pharmacologic therapy for acute tubular injury; mostly, we're providing supportive care, keeping the blood pressure up, optimizing the volume status, and using dialysis where appropriate.

In patients with hepatorenal syndrome, we treat with a combination of IV albumin and vasoconstrictors with the idea that we're going to reverse the splanchnic vasodilatation that's causing that cascade of neurohormonal responses that are ultimately leading to renal vasoconstriction. So, by reversing the splanchnic vasodilatation, and we ultimately improve renal profusion and improve kidney function.

Dr. McDonough:
How does it impact transplant evaluation?

Dr. Reau:
Yeah. Transplants challenging because every patient is ranked based on a score, their MELD score, and the worst their MELD, the more likely they are to become transplanted. And so, if a person is listed for transplant or is very likely to need a transplant, is going to be a good candidate. We're a little less aggressive in the patients that are very advanced because they're going to go to transplant quickly. So, we don't ever want to neglect a patient or not reverse something that is reversible. But if a person comes in and their creatinine is 5 or 6, and their MELD is 38 or 39, then interventions are less likely to improve the kidney function. That patient is much more likely to go transplant very soon.

It's more controversial if you have someone that comes in in between. And here, it's important to remember that if you address the hepatorenal syndrome, if you make the kidney healthier, that's going to make the post-transplant course much better. You're in better renal function after transplant, and that's really impactful for the patient. And so, if you have hepatorenal syndrome that is reversible, we absolutely want to address that prior to transplant, recognizing that, yes, your MELD might drop a little bit but not so substantial that it's worth trying to compromise the kidney.

Dr. McDonough:
Now, Dr. Reau, looking specifically at decompensated cirrhosis, we know that it impacts multiple organs. Based on that, how can teams work together to make treatment decisions?

Dr. Reau:
As liver disease becomes more advanced, our patients do end up with multiple organ failures, and it's easiest to have multidisciplinary discussion when your patients in the intensive care unit. So much that many academic or probably most academic centers actually have multidisciplinary rounds where we all sit together and go through the plan on that patient because we understand that working together is so vital.

It's a little more fractured when you're trying to manage a patient either as an outpatient or on the floor because now, you're working with multiple consulting teams. But I think it's important to recognize that it is a integrated care that's best for the patient. And having algorithms that allow you to have consensus and highlight good decision-making points like this is how you treat something, and if it doesn't get better, then these are the teams that now get together to understand the next step; that's vital. Otherwise, you're going to miss opportunity to make your patient better.

Dr. McDonough:
Dr. Regner, how much is established by protocols versus how much is team decision-making?

Dr. Regner:
So, if we take just the approach to acute kidney injury in patients with cirrhosis, is an example of an established protocol. If we look at the AASLDs and recommendations on how to approach this, it's a very algorithmic approach where we identify AKI. We perform a diagnostic evaluation. We stage AKI. We perform some risk-factor management and then consider giving fluids. It's all very protocol-driven, and the nice thing about that is that everybody on the team can see that protocol and be aligned on trying to implement that protocol in the care of these patients earlier on in the course of their disease.

Similarly, if we used vasoconstrictors and albumin to treat HRS, there are established protocols from that when we initiate therapy and how we monitor therapy. But every patient is an individual, and how they respond to each of these different interventions really requires the team to be paying close attention to them, taking a nuanced approach, and then reevaluating their approach on a daily basis. And so, the nephrologist has to be talking with the hepatologist and the transplant surgeons, making sure that we're all really aligned.

Dr. McDonough:
As we approach the end of our program, I have a final question for you, Dr. Reau. How do you approach AKI in a person who has had TIPS?

Dr. Reau:
Yeah. That's a great question because, in theory, a TIPS placement, which connects the portal venous flow to the venous outflow, should eliminate the pathophysiology of portal hypertension. Now, in actuality, it only bypasses a small portion and can have certainly TIPS dysfunction. So, the TIPS might not have been small, might have been a caliber that didn't get rid of all portal hypertension, or you could have a clog or a stenosis, or the TIPS could have failed. And so, when you look at a patient who has had a TIPS placed, you want to be a little hesitant before you label them as hepatorenal syndrome. But they certainly can still have HRS.

The flip to that is also a person who needs to have an urgent TIPS placed. So, you have refractory variceal bleeding, or you have a person who's really struggling with a hydrothorax, refractory ascites, where TIPS is clearly indicated. And in those patients, because of the interventions and the pathophys, they are at risk for kidney injury, but acute kidney injury is not always a strict contraindication. It's a cautionary tale, but it's not always a strict contraindication to TIPS in that patient.

Dr. McDonough:
So, if you have a patient who has HRS is TIPS an approach you'd consider?

Dr. Reau:
I don't think that we would place a TIPS as a treatment for HRS. I think it's more a patient who has HRS that might have another indication for TIPS. And HRS, again, is a spectrum, so you would not think about placing a TIPS unless it's life-threatening and someone who has very advanced stage 3 HRS where you know the kidneys are going to struggle.

Dr. McDonough:
That's a great way to round out our discussion, and I want to thank my guests for their insights into the management of acute kidney injury in cirrhosis.

Dr. Reau, Dr. Regner, it was great speaking with you both today.

Dr. Reau:
Thank you so much for inviting me.

Dr. Regner:
Thank you. Great discussion.

Announcer:
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