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Treatment of Overt Hepatic Encephalopathy

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Dr. Brown:
This is CE on ReachMD, and I'm Dr. Robert Brown from Weill Cornell in New York. Here with me today is Dr. Steven Flamm from Rush University in Chicago.

Steve, we've talked about the treatment of minimal HE, what can you tell us about treatment for overt hepatic encephalopathy, or overt HE?

Dr. Flamm:
Thank you, Bob. This is a very important question. If providers care for cirrhotic patients, there will be cirrhotic patients that develop, within the course of their disease, overt encephalopathy, which means symptoms related to diminished cognition that patients have off and on in the setting of their disease course related to cirrhosis. And it's very important for providers, even if they are not the ones who are primarily treating the HE, to identify it and make sure the providers are sending those patients to GI or liver doctors that can help with patients that have these symptoms.

Now, when you're looking at a patient with HE, you need to diagnose it first, and patients who have overt presentations, they have behavioral changes, personality changes, disorders. They have confusion, slurred speech, so it shouldn't be missed. And when you have such a patient, how the GI/liver provider certainly will treat it is they will look at 2 different things.

Number one, what is the provocation? What is happening that causes the encephalopathy? Almost always, there's a reason for it. That can relate to dehydration from diuretics. It can relate to overmedications with medicines that have psychoactive effects that many liver patients are on. It can relate to infections like urinary tract infections, cellulitis, or spontaneous bacterial parotitis, or it can relate to upper GI bleeding. So there are a lot of provocations, Bob, that cause HE.

And then secondly, we need to use ammonia-lowering agents in the context of HE, and that includes medicines like lactulose or rifaximin. Very important. You will help the patients get better from their episode if you correct provocations and put patients on rifaximin. You will lessen the risk that they have recurrent episodes of overt encephalopathy. And when you do that, you will decrease their hospitalizations, you will increase their quality of life, and overall help their disease course in a more favorable way.

What do you do in practice, Bob?

Dr. Brown:
Well, I think you've highlighted a couple really key points. The first is, when someone has HE that worsens, we have to look for the precipitants. Most of them will be precipitated by something not spontaneous, and that would be either infection, dehydration, or bleeding, and to take a careful medication history, including over-the-counter medications that may be sedating and patients may not report.

And then we have to initiate first-line therapy, lactulose, to reverse the acute decompensation, but then rifaximin plus lactulose to prevent recurrence.

But I think you highlighted an important thing. You said we use ammonia-lowering pharmacotherapy. What role does ammonia have in making the diagnosis or the treatment?

Dr. Flamm:
That's also a great question, Bob. Despite the fact that we use what we call ammonia-lowering agents, the ammonia level itself is not very predictive of whether or not a patient has encephalopathy, and you don't treat the level. We all like numbers today, we like to treat numbers in labs, but you don't use ammonia levels for treatment, and you certainly don't use it for diagnosis either. And yet, ammonia seems to have some contribution to the clinical presentation and other toxins that are in the blood that we don't measure in patients with portal hypertension and cirrhosis. So we use ammonia-lowering agents, Bob, but you don't really follow the levels.

Dr. Brown:
So ammonia-lowering therapy, but don't measure the ammonia.

So our time is up, short, sweet, and ready for practice. Thanks for listening.

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