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Hepatic Encephalopathy: More Common Than You Think

Announcer:
Welcome to CE on ReachMD. This activity is provided by TotalCME and is part of our MinuteCE curriculum.

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Dr. Jesudian:
This is CE on ReachMD, and I'm Dr. Arun Jesudian. What is the burden of disease with hepatic encephalopathy?

First, let's start by talking about cirrhosis, which is the final stage of scarring or liver damage resulting from any chronic liver disease. Common ones include alcohol-associated liver disease, or metabolic dysfunction-associated steatotic liver disease, or what we commonly refer to as fatty liver, in addition to hepatitis B and C and a number of other rarer causes of chronic liver disease.

Cirrhosis is highly prevalent in the United States, and its incidence is increasing in the US population. Early on, patients with cirrhosis have compensated disease, and what I mean by that is they do not have any symptoms or major complications of cirrhosis, although they do have stage 4 scarring or fibrosis in their liver. Patients with compensated cirrhosis are expected to live 12 to 15 years, whereas patients with decompensated cirrhosis, who have developed a major complication, are much sicker and might not survive 2 years without a liver transplant.

Complications of cirrhosis that lead to decompensation include ascites—fluid accumulating in the abdomen—bleeding from varices, and hepatic encephalopathy. And hepatic encephalopathy is really the most burdensome of those complications. Hepatic encephalopathy impairs cognition and mental status in affected patients, and it can really affect their quality of life, their ability to complete activities of daily living, their ability to drive safely or work. It can impair their sleep cycles. It can even affect their mood. And most of these patients require caregivers, and these caregivers can burn out because of the burden placed on them.

Hepatic encephalopathy occurs as a consequence of liver insufficiency and portosystemic shunting in the setting of cirrhosis. Liver insufficiency means that there are less healthy hepatocytes, or liver cells, as they get replaced by scar tissue or fibrosis.

And hepatocytes have many functions. One of the major functions is to remove toxins from the bloodstream, toxins like ammonia that are generated by bacteria in the intestine, primarily the large intestine, the colon. Ammonia is a byproduct of ammonia breaking down proteins there. When the liver cannot remove ammonia from the bloodstream in the setting of liver insufficiency, that ammonia can travel up to the brain and cause the altered mental status, the impaired neurotransmission that's characteristic of hepatic encephalopathy.

Portosystemic shunting means that blood flows around the liver in the setting of cirrhosis and portal hypertension. A liver with cirrhosis is shrunken down and lumpy, bumpy, and nodular from all that scar tissue, and blood from the intestines cannot properly flow through it on

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the way up to the heart. So some blood finds ways to flow around the liver through portosystemic shunts, which means that ammonia-rich blood is then reaching the brain and contributing to hepatic encephalopathy.

So what might tip you off to a patient having cirrhosis and portal hypertension and being at risk of hepatic encephalopathy? One major clue, in addition to the presence of chronic liver disease, is the platelet count. Lower-than-normal platelets, or thrombocytopenia, can tip you off to the presence of cirrhosis and portal hypertension, because as blood flow backs up into the abdomen, your liver's neighbor, the spleen, gets enlarged. And enlarged, engorged spleens have a tendency to consume or chew up platelets, leading to a lower-than-normal platelet count.

If you take one thing away, be aware of the prevalence of undiagnosed cirrhosis and increase your index of suspicion in patients with subtle alterations in cognitive function.

And now you know. Thanks for listening.

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