#### SOMETHING FOR THE PAIN

EPISODE 22: Surveying Substance Use Disorder: Alcohol

(30 mins)

(0:00)

[cue guitar music]

[Sam Steffen]

This is *Something for the Pain*, a podcast produced by Project ECHO Idaho, made for Idaho's healthcare professionals working to prevent, treat, and facilitate recovery from opioid and substance use disorders throughout the Gem State. I'm your host, Sam Steffen.

[theme song]

Today we're kicking off season three of our podcast. If you're just joining us, season one of *Something for the Pain* was all about opioid prevention, treatment and recovery in Valley County and throughout Idaho. For season two, we honed in on substance use disorder as it affects pregnant patients, and reviewed highlights from ECHO Idaho's former Perinatal Substance Use Disorder series.

For our third season, we're going to be zooming out a little bit to talk more generally about a few of the substances commonly associated with substance use disorders, specifically: alcohol, marijuana, kratom, psilocybin, opioids, fentanyl, methamphetamines, and benzodiazepines. Each episode in this season will focus on one of these substances to provide evidence-based insights and updates from an interdisciplinary team of Idaho healthcare experts, about the latest research, common health conditions associated with chronic and long-term use of each, as well as treatment options and best clinical practices that translate to the primary care setting.

Today's episode features a presentation by Dr. Elsbeth Jensen-Otsu, hepatologist and gastroenterologist at the Boise VA Medical Center, on the topic of "Alcohol-Related Liver Disease." While Dr. Jensen-Otsu is employed by the Boise VA Medical Center, she would like listeners to know that her opinions are expressly her own and do not represent those of the Boise VA Medical Center. This lecture was recorded on February 28, 2022 as a part of ECHO Idaho's Viral Hepatitis and Liver Care series. Here to introduce today's presenter is ECHO Idaho's Viral Hepatitis and Liver Care series co-facilitator and panelist, Dr. Abby Davids.

[Abby Davids]

And I'm Abby Davids, I'm one of the faculty at the Family Medicine Residency of Idaho, Boise and I'm the director of our HIV and Viral Hepatitis Fellowship. So thank you all for joining us, we're really excited today, Dr. Jensen-Otsu is going to give us a talk on "Alcohol-Related Liver Disease," so, Elsbeth, I'll hand it over to you!

[Elsbeth Jensen-Otsu]

Hi, this is Elsbeth Jensen-Otsu, I'm a GI and hepatology doctor at the Boise VA medical center. We're going to talk about alcohol-related liver disease, something that I see super commonly in my clinic. So

the learning objectives for today: one is to understand the spectrum and stages of alcohol-associated liver disease; second is to be able to identify patients who are at risk for ALD, and then third to kind of understand the basic management for alcoholic hepatitis and alcohol-associated cirrhosis.

So this is what we're going to talk about as far as outlines, we'll talk about the spectrum of ALD, alcohol use and biomarkers that can be useful to clue you in that someone may be using alcohol excessively and affecting their liver function and other things. And then we'll talk about alcoholic hepatitis and associated cirrhosis and then a little bit about liver transplant for ALD—this is something I get asked about all the time, so just kind of talk about the basic stuff that's good to know from a referring provider's perspective.

So kind of jumping in to talking about the spectrum of ALD: Alcohol associated liver disease is super common. It's estimated that 2% of all the US population has some form of alcohol associated liver disease, but it's a very broad spectrum, and multiple stages can exist simultaneously. So this is one of the challenging things that comes up is, you know, do people have cirrhosis? Do they have just alcoholic hepatitis or steatosis? And people can actually kind of move back and forth between these stages, which is a little bit unique to alcohol associated liver disease as opposed to other chronic liver disease, where it's more of a one-way street. And again, multiple stages can exist simultaneously, so more than 50% of people who present with clinically apparent alcoholic hepatitis actually have underlying cirrhosis as well. You know, after two weeks of more than 60 grams of alcohol a day, 90% of people will get steatosis, which just means excessive fat in the liver, but this is reversible after 4-6 weeks of abstinence. However, with ongoing heavy alcohol use, it can progress to what we call steatohepatitis which just means that there's, in addition to fat in the liver, also inflammation, and that's what can lead to fibrosis and then eventually cirrhosis and complications of cirrhosis, including decompensated cirrhosis and hepatocellular carcinoma. So it's not necessarily a guarantee that if someone drinks excessively they will develop cirrhosis but you know even if people get significant fibrosis, there's still a bi-directional arrow to just simple steatosis so with prolonged alcohol abstinence, even with scar tissue, can have regression back to a relatively normal looking liver.

So alcohol use and bio-markers: alcohol use is increasing. Overall use has increased significantly over the last couple decades and alcohol use disorder is estimated to have increased by 50% between 2001 and 2013. We define alcohol use disorder as escalating alcohol consumption despite attempts to cut back, personal consequences or alcohol cravings. In the US and a lot of other places around the world AUD is increasing, so universal screening for alcohol use disorder is recommended. I will say anecdotally, and there's been some data to support this, too, now that we're a couple years into this pandemic, but that hospitalizations for alcoholic hepatitis and alcohol related liver disease have increased substantially during COVID. People are drinking way more than they used to. So the universal screening, initial recommended screening question is "How many times in the past year have you had 5 or more drinks in a day?" for men and "4 or more drinks in a day?" for women. And if that screen's positive, then it's recommended that you ask them more detailed questions about alcohol use.

So how much alcohol is too much? This is often really confusing for patients because, you know, we all know people who drink way more than this and who never get alcohol related liver disease, they never get alcoholic hepatitis or cirrhosis, but the risk of cirrhosis actually significantly increases with more than 40-80 grams a day for men or 20-40 grams for women, and that's not that much. So the

recommendation actually is less than or equal to 2 drinks a day for men and less than or equal to 1 drink a day for women, and zero if you have any underlying liver disease.

Other risk factors for developing alcoholic related liver disease include: the pattern of alcohol drinking, so daily drinking, drinking while fasting, and then also heavy binge drinking carry increased risk, as well as tobacco use, increased BMI, which increases your risk for steatosis in the liver that's not related to alcohol, and then of course if you have other chronic liver disease such as viral hepatitis, hemochromatosis, et cetera.

So if someone has underlying liver disease, I tell them there's really not a safe level of alcohol they can consume without increasing their risk of progressing to a more advanced stage of liver disease than where they're at. There's some data that suggests maybe that the kind of alcohol consumed makes a difference, you know, like maybe potentially wine may be better than other kinds of alcohol, but it's really not very strong data.

So what are some signs that there may be alcohol associated liver injury or excessive alcohol consumption? Well, there's both indirect and direct bio-markers that can be useful. So for indirect markers, obviously LFTs.

### [Sam Steffen]

For those who may not be familiar with this term, an LFT is an abbreviation for a liver function test. These tests are also sometimes referred to as liver panels and involve blood tests that measure enzymes, proteins and other substances that are produced by the liver. You may also hear Dr. Jensen-Otsu refer to ASTs and ALTs; an AST is a blood test that measures the Aspartate Aminotransferase enzyme; an ALT is a blood test that measures the levels of the Alanine Aminotransferase enzyme. These are enzymes produced in the liver that show up in higher quantities in the blood when the liver has been damaged.

### [Elsbeth Jensen-Otsu]

So typically we see an AST/ALT ratio of 2:1 with someone who's having liver injury related to alcohol. You know, not everyone reads a textbook, but typically the AST will be greater than the ALT if not in that classic sort of 2:1 ratio. Also macrocytosis (enlarged red blood cells) and thrombocytopenia (low blood platelet count) are also common because of the effects of alcohol on the bone marrow. So if you see someone who has, you know, both of those signs, I would definitely ask them about alcohol use. Ferratin can also be really elevated in alcohol use. It can also be elevated in chronic liver disease in general, but it's pretty frequently that I get referred to someone for quote/unquote hemochromatosis when really the ferratin is related to excessive alcohol consumption.

So there's also direct bio-markers that you can use to detect alcohol use. These probably are not going to be ordered very frequently from primary care, but I thought it was just kind of useful to mention. So you have the serum alcohol level, which typically will detect alcohol use within the previous hours. Urinary ethyl glucuronide, which can detect alcohol in the last like 2-3 days with a fairly good positive predictive value, and then there's a new test called a PEth test which we use frequently in the pre-liver transplant world. It has a half-life of about 10-14 days and what it is, is, it's a phospholipid that's formed by the reaction of phosphatidylcoline with ethanol and it forms on the outside of the red blood cell. So you can use it to detect alcohol within actually weeks prior to the testing. So a level of more than 20 has

a positive predictive value of 85% for alcohol use and of greater than 80 it's fairly sensitive for more than 4 drinks a day.

So moving on to alcoholic hepatitis and alcohol associated cirrhosis. So starting with alcoholic hepatitis. What we mean when we say alcoholic hepatitis is basically the acute onset of symptomatic hepatitis which usually presents as jaundice. It occurs in about 20% of heavy drinkers and it's a very common reason for hospital admission, which has been increasing like I said over the last decade or so. The reason I think it's important to know about even if you're primarily taking care of folks as an outpatient is that it carries with it a very high mortality rate with severe alcoholic hepatitis. So 28 day survival with severe alcoholic hepatitis, which typically we define as a discriminant function greater than 32, which, we'll talk about. You know, some studies will quote the mortality associated with that to be up to 50% which I mean, if you think about, that's up there with like STEMIs (ST-elevation myocardial infarction) and other very severe acute reasons for hospitalization.

So I have a kind of typical case just to get our minds working about how these people might present.

## [Sam Steffen]

Just a word here about this patient case. Dr. Jensen-Otsu is presenting a hypothetical case here, but it's typical during ECHO sessions to hear real de-identified patient case presentations, delivered from practicing Idaho clinicians, physicians, social workers and other members of patient care teams. If you're a practicing clinician or physician, you can present your de-identified case to an interdisciplinary expert panel to help others learn and to receive free expert feedback from an interdisciplinary panel. Check out ECHO Idaho's website for more details. www.uidaho.edu/ECHO

#### [Elsbeth Jensen-Otsu]

So this is a 43 year old man with a history of alcohol use disorder who presents with jaundice and right upper quadrant pain. And then I put kind of pretty typical labs that we see: white blood cell count of 14 which is a little bit elevated, normal hematocrit, platelets 140 which is a little bit low, an AST and ALT that are both elevated but AST greater than ALT, and then a total bilirubin of 8, which is, you know, definitely abnormal. Normal kidney function but an INR (international normalized ratio) of 1.7 which is also elevated, although not hugely hugely elevated, but definitely abnormal. So like I mentioned, the typical clinical presentation includes jaundice. A lot of these folks will have tender hepatomegaly (enlarged liver), so even though we say the liver doesn't hurt in liver clinic, the liver capsule actually is enervated, so when there's a lot of swelling from a very acute inflammatory process in the liver, then you can get tender hepatomegaly. It's not typical, it's not usually as bad as like acute cholecystitis (inflamed gall bladder) but you can have a little bit of tenderness in the right upper quadrant with alcoholic hepatitis. These folks may or may not have stigmata chronic underlying liver disease like spider angioma (enlarged blood vessels), palmar erythema. If present, it usually indicates underlying cirrhosis. And they commonly do also have a leukocytosis and AKI and unfortunately some of them do progress to multi-system organ failure.

So, what should you order next? So, a 43-year-old gentleman presenting with jaundice and mild right upper quadrant pain...?

[lan Troesoyer]

I think the liver ultrasound would be helpful.

[Sam Steffen]

Speaking here is Ian Troesoyer, Nurse Practitioner at Bear Lake Community Health Centers in Logan, Utah, and a regular participant at ECHO Idaho.

[Elsbeth Jensen-Otsu]

Absolutely, yes. You definitely want to get an ultrasound. The big thing, you know, I told you this person has alcoholic hepatitis, but the big thing you want to make sure is that there's not something else going on. I mean, just because he has a history of alcohol use disorder doesn't mean he isn't also entitled to have some other kind of liver injury, right? So, a right-upper-quadrant ultrasound, to make sure there's not like a biliary issue going on, you know, like a gall stone, and then viral hepatitis serologies, HIV, again, to rule out other causes of acute liver injury. And then, because these people are at such high risk for getting infections that complicate their hospitalization, I would also usually get blood cultures, urine cultures, and then a chest x-ray on admission, too.

So his ultrasound came back with an enlarged liver that appears echogenic. Some nodularity of the edge of the liver, which you know may indicate cirrhosis. He also had splenomegaly but no ascites, and the bile ducts were normal. Otherwise, everything looks okay, and his alcohol history is, he drinks about 4-6 beers a night, but he recently increased to add you know like 4 shots with that as well. Which is a pretty typical, I would say, story for someone that we see in the hospital with alcoholic hepatitis, is a chronic history of excessive alcohol intake with a recent increase, but not everybody necessarily has that. So just because you don't have a recent increase doesn't mean that you don't have alcoholic hepatitis.

So there's a couple scoring systems that are good to know about that we use to estimate short-term survival and determine treatment for alcoholic hepatitis. So one is called the Maddrey's Discriminant Function, and it includes bilirubin, INR, those are the two biggest contributors. And a score of greater than 32 is associated with a higher risk of death. So that's usually what's used for the cutoff for steroid administration, but you can also use the MELD (Model for End-Stage Liver Disease) Score, too, which includes bilirubin, INR, creatinine and sodium, and it's just sort of a global sort of assessment of liver function.

[Sam Steffen]

The MELD score that Dr. Jensen-Otsu is referring to here, spelled M-E-L-D, is an abbreviation that stands for Model for End-Stage Liver Disease.

[Elsbeth Jensen-Otsu]

And there may be sort of a sweet-spot for steroids, with a MELD of between like 25 and 39. So people who are like way way high, with a MELD score of like 50, probably don't benefit much from steroids, just because they're too sick.

[lan Troesoyer]

Can you talk about the physiological benefits of steroids in these patients, why that helps?

[Elsbeth Jensen-Otsu]

Yeah, so the idea behind steroids is that they're anti-inflammatory, because it's the acute inflammatory reaction to alcohol and the toxic metabolites from alcohol that cause the hepatitis. And so the theory behind steroids is that if you decrease the inflammation, then that will decrease the amount of injury that's causing the damage to the liver, right? The initial trial was looking at steroids in alcoholic hepatitis from like the early 90s showed a significant benefit, like a 40% increase in survival, which is huge, right? But there's been more recent studies, specifically this study called the STOPAH Trial, this is from *The New England Journal* from 2015, that did not show much benefit, so, you know, there's some debate I think as to whether or not steroids are really effective treatment for alcoholic hepatitis. You know, proponents of steroids would say that the patient population in the older trials versus the newer randomized control trial from 2015, that the patient population was a lot different but you know I think there's still some uncertainty. The other sort of argument against steroids is that yes, it may help like short-term mortality within 30 days but no one's ever shown that it helps 60 or 90 day mortality. But considering how sick these people are, I do recommend steroids if someone falls within the severe alcoholic hepatitis.

But the other things that are actually probably equally if not arguably more important are nutrition and alcohol abstinence, which I think are both severely underemphasized in treating patients with alcoholic hepatitis. This is an older study, but I quote it to patients all the time because it's really telling.

# [Sam Steffen]

The study Dr. Jensen-Otsu is referring to here is one titled "Indication of liver transplantation in severe alcoholic liver cirrhosis: quantitative evaluation and optimal timing." This is a study that was conducted in France and issued in the Journal of Hepatology in 2002, lead by Bartholomeus Johannes Veldt.

### [Elsbeth Jensen-Otsu]

So for people with severe alcoholic hepatitis, they followed these folks two years out and for people who stopped drinking, most people actually did survive. So, you know, in this study up to 75%, which I know is a little conflicting with some of the other data that I've quoted to you, but for the people who went back to excessive drinking—basically they were all dead at two years. So getting people into effective substance use treatment is way more important than whether or not they get steroids. I mean, that's hugely impactful in these people's short- and long-term survival.

The other thing that's actually super important is nutrition. So a lot of these patients who get admitted are severely malnourished. And even if they're sedentary, they're all hypermetabolic because of the liver injury, so the recommendation actually is that they be on at least a 2500 kilo-calorie per day diet, and a diet that's high in protein. So this is a study comparing steroids which at the time was the standard of care to enteral nutrition through like a naso-gastric tube.

## [Sam Steffen]

Dr. Jensen-Otsu is referring here to a 2006 study conducted by Cabre et al. titled "ESPEN Guidelines on Enteral Nutrition: Liver Disease"

### [Elsbeth Jensen-Otsu]

And it wasn't statistically significant, but the patients who got a feeding tube did better than the patients who got steroids. So I think alcohol abstinence, nutrition, those are things that are no-risk—

unlike steroids which you know potentially have some risk as far as bleeding and infection and things like that—and may be more effective if not more effective than steroids.

Moving on to alcohol-associate cirrhosis. So excessive alcohol is a very common cause of cirrhosis. You know generally it's a clinical diagnosis and we don't biopsy, but there are some kind of diagnostic challenges that can come up with alcohol-associated cirrhosis. So, one of them is folks who maybe drink a little bit more than they should, but they also have a lot of underlying risk factors for non-alcoholic fatty liver, so we get into this debate of like, is it ASH—alcoholic steatohepatitis—or is it NASH?—Non-Alcoholic steatohepatitis? I mean, in the end, if they have cirrhosis, it doesn't really matter because you're going to tell them to cut out all alcohol. It only really matters in the setting of if someone needs a transplant, how much substance use treatment or counseling they need before you would consider referring them or listing them for transplant, but it is something that comes up a lot. The other thing is that it can be really difficult to identify cirrhosis versus just alcohol related liver disease (and not cirrhosis) because the non-invasive markers that we use are increased with active alcohol use. So Fibro scan or ultrasound elastography will be increased with active alcohol use because the swelling of the liver that's associated with those actually will increase your Fibro scan results. So I typically actually wait about 3 months after people have been off alcohol in order to do a Fibro scan cause otherwise you're going to get a high number and you're not going to know if it's real or not. The other thing is, the things that we calculate, just lab-wise, like the fib-4 and the APRI, they can also over-estimate cirrhosis in someone with chronic alcohol use because they all include platelet and AST which are...you know, the platelets could be low from alcohol use even in the absence of cirrhosis, as can AST. So that can be challenging.

So how do you identify people with cirrhosis? So what I do is a physical exam. So if they have signs of chronic liver disease like spider angiomata, palmar erythema then that would be more suggestive of cirrhosis. And then the other thing is looking at their liver imaging. So if they have a big huge fatty liver, then you could say, "Well, there could be underlying cirrhosis there but we can't really say." But if their liver is really small and nodular, then they probably have cirrhosis. And then you know ultimately the test of time is helpful, you know if people stop drinking and their labs all go back to normal and their ultrasound looks fine and you Fibro scan them and it's normal, then it's fairly clear-cut. If after 3-6 months all those things haven't changed and you re-image and their liver looks small and shrunken and nodular, then they probably have cirrhosis. But it can be, at the time, it can be very difficult, if you're seeing someone kind of early in their abstinent period.

As far as management of alcohol associate cirrhosis, you know, pretty standard as far as what we would recommend for anyone with underlying cirrhosis, so hepatocellular carcinoma screening, variceal screening, if indicated, managing any complications and then of course nutrition is still important in these folks as well. And then, alcohol avoidance. You know there's not a lot of medications that have been studied for alcohol use disorder in people with chronic alcoholic liver disease, but there are still things that you can use and I mean, I get asked a lot about the safety of Naltrexone in these folks and it hasn't been directly studied, but it's still probably safer than a significant alcohol relapse.

And then the other thing I wanted to mention, just kind of going back to the idea of stages and sort of reversibility: alcohol associated cirrhosis is by far the most common thing where we see people who look like they are on death's door with decompensated cirrhosis, and they actually can recover a significant liver function, to the point where they actually recompensate. So you know we see people who are jaundiced with ascites and everything and then with prolonged alcohol abstinence, their liver

function can return to—not 100% normal, typically—but to the point where at least their labs look okay and they don't have ascites or jaundice or anything like that.

So just a little bit about liver transplant for alcohol associated liver disease. ALD is now actually the leading indication for liver transplant in the US, more so than hepatitis C, which is a recent shift over the last few years. Probably NASH is going to be the number one at some point, but for now at least alcohol is the number one. You know the timing of the referral is something I get asked a lot about. Some centers will consider liver transplant prior to six months of abstinence, but I would think about if someone hasn't improved after 3-6 months to at least start thinking about that. The reason for this is kind of two-fold. So, one is that abstinence for less than 6 months is associated with a higher risk of relapse post-transplant. The other is just what we were talking about before which is that, you know, a lot of these people will recover to the point where they don't need a liver transplant. So, you know being evaluated for a liver transplant is a big deal, it's a lot of medical visits, it's a lot of burden on the patient. So if they don't need a liver transplant, you don't really want a liver transplant. But generally speaking, if their liver function has not recovered and their MELD is still in the high teens, low twenties, or if they have child C cirrhosis, then we think about referring for a transplant. Every transplant center requires people to be engaged with substance use treatment and have either completed or in the process of completing a relapse prevention program. There are some places that will consider transplant for severe alcoholic hepatitis, but it's pretty controversial. You know part of the reason that there's such this rigorous screening process is that unfortunately a fair number of people do have a relapse after transplant. One study I found suggested even up to a quarter of folks will have a significant relapse after transplant and the predictors of relapse are: abstinence less than 6 months, psychiatric comorbidities, being unmarried and then smoking. Liver transplants are a very scarce resource and people have to be screened very heavily, and it's something that's very guarded to try to maximize benefit for the greater good, but it does get to be dicey sometimes in some situations. There are some places that will consider folks for transplant who have severe alcoholic hepatitis but it's a pretty low number of centers and it has to be someone who had never been told that they had liver disease or had been told that they should stop drinking before. So typically these are people who are very young who are very, very sick and they have to sign all these contracts about that they'll go to treatment afterwards, they have to have very strong social support and no other psycho-social risk factors. So it's a very select group of people, but there are some places that are considering doing transplants for first episode severe alcoholic hepatitis.

So some general contraindications for liver transplant, and some of these are relative, but: a lack of social support is an absolute contraindication. So people really need to have good social support to help them through the process. Ongoing alcohol or substance use. Significant medical comorbidities. So if you have someone who has like heart failure or like super uncontrolled diabetes, that's a contraindication. Age is sort of a relative contraindication, but liver transplant is a really really big surgery, so it just gets more and more difficult to recover after age 70. And then I put BMI on here, I think that's a relative contraindication and varies by center, and there's a lot of folks who think there should not be a BMI cutoff, but there's some surgeons who worry about like wound healing and stuff like that.

So key points for today: alcohol associated liver disease, very common and unfortunately increasing in prevalence. Some risk factors we talked about: excessive alcohol use, elevated BMI. Women are particularly at risk because of a lower threshold for alcohol use that puts them at risk for cirrhosis, and then of course folks who have underlying liver disease otherwise. There's universal recommended screening for alcohol use and, you know, look for indirect markers of excessive alcohol consumption in

your patients like really high MCV (mean corpuscular volume) or low platelet count and of course their liver function tests. Alcohol abstinence and nutrition are really key for management of both alcoholic hepatitis and cirrhosis, and with abstinence, liver function can recover even if underlying cirrhosis persists. The liver is an amazing organ in that it has regenerative capacity. So even if someone is decompensated, it doesn't necessarily mean that they're always going to stay that way if they're able to achieve long-term abstinence.

# [Abby Davids]

That was great, Elsbeth. Thank you so much. I have a question about medications for alcohol use disorder, if you don't mind talking from a liver expert perspective, which ones you tend to use, which ones tend to work the best, and maybe Winslow can chime in, too?

### [Elsbeth Jensen-Otsu]

Yeah, so I don't have a lot of you know personal experience prescribing these medications, but you know looking at the data there's some risk of liver-related toxicity with Naltrexone, but the problem is none of these have really been studied in anyone with chronic liver disease, right? The only one I think is Baclofen? Which, my understanding from our substance use treatment folks here at the VA, they don't think is very effective. And Winslow probably can maybe weigh in more. So the ones that I have seen used more recently are gabapentin and Naltrexone. Now, I would be somewhat hesitant to give someone Naltrexone if their LFTs were still markedly abnormal from alcoholic hepatitis, but I think if they're getting better...you know it's still probably safer than significant alcohol relapse. Basically, anything is better than nothing, you know, that's kind of the take-home. There was a big population-based study from Massachusetts that I saw looking at folks with alcohol use disorder and progression of liver disease and treatment with anything was better than nothing, and most things appear to be safe but you have to monitor them, I think, is kind of the take-home. Winslow, do you want to weigh in?

### [Winslow Gerrish]

Uh, yeah, what you said are all things that I would agree with...

#### [Sam Steffen]

Speaking here is Winslow Gerrish, Clinical Psychologist and Director of Behavioral Sciences, Research, and Grants at Full Circle Health, Boise. He's also one of the standing panelists for ECHO Idaho's Viral Hepatitis and Liver Care series.

## [Winslow Gerrish]

I don't know if there's data to support this but what I've seen is Naltrexone for reducing heavy drinking, so that initial reduction, and reducing cravings, and then Acamprosate for helping maintain abstinence. And I believe Acamprosate is a little easier on the liver, or on liver function. And then the others that you mentioned with the same caveats.

### [Elsbeth Jensen-Otsu]

Baclofen has been specifically studied in folks with alcoholic related liver disease and thought to be safe. Naltrexone, gabapentin, Topiramate, Acamprosate, basically no studies. And there's more concern about Naltrexone, but if you look on *Liver Tox* which is an NIH resource looking at drug-induced liver

injuries for medications, basically everything is going to show up on that. So I think that there's maybe a slightly higher risk with Naltrexone, but the risk is probably, it sounds like, been overestimated and is probably safer than a significant alcohol relapse.

I'm seeing a question about coffee? Um, no, yeah, so coffee has actually been shown to be protective for all kinds of liver injury and chronic liver disease. It's actually a fair amount of coffee that you have to drink in order for those stats to become statistically significant, but coffee's not going to hurt your liver and potentially may be good.

### [Winslow Gerrish]

And then I have one kind of selfish question just because it's come up for me with a couple patients the last few months, is being on the liver transplant list and using marijuana and kind of what you've seen with that? Cause for me I've had two patients in the last couple months where that is something that they do not want to stop although they have been able to stop alcohol use.

### [Elsbeth Jensen-Otsu]

Yeah, I think that's center-dependent, but typically I would say most places are going to say no marijuana. The hard thing about marijuana is that you can test positive for marijuana even though you're not actively using because it can get stored in your adipose tissue, and so I've seen folks sort of get put on hold or even delisted in settings where it really wasn't clear someone was really actively using. But yeah, I would say typically that's a no-no for transplants. But it's hard particularly in places where it's legal, right? Like, you know, half of the US, now, but...yeah, kind of the same thing with tobacco, like tobacco is legal but it's still a big no-no for transplant. And you could argue probably that tobacco's kind of a bigger problem from like a wound-healing, surgical-risk, clot-risk, those kinds of things, than marijuana, but transplant centers don't want to see anything that's even remotely considered illicit. And I think the reason is that most of the people who get listed for liver transplant end up dying on the waiting list, so there's a moral obligation from the transplant center's perspective to try to make sure that whoever does get a liver transplant is going to actually benefit from that from the greater good standpoint. I prefer being on the referring side and not the transplant side, because they have to make sometimes some really tough decisions that feel really morally sort of gray, I think.

#### music

### [Sam Steffen]

That again was Elsbeth Jensen-Otsu, hepatologist and gastroenterologist at the Boise VA Medical center presenting "Alcohol-Related Liver Disease." That lecture was recorded live on Feb. 28, 2022 as a part of ECHO Idaho's Viral Hepatitis and Liver Care series.

If you'd like to watch the Zoom recording of that presentation, that video is currently available on the ECHO Idaho YouTube channel, which you can access through our website. The Powerpoint slide deck as well as information about how to contact some of the organizations and services mentioned in that talk, are available in our podcast show notes, on our podcast webpage: <a href="www.uidaho.edu/echo-podcast">www.uidaho.edu/echo-podcast</a>

### Banjo music

If you're interested in joining our free, live ECHO sessions to receive Continuing Education credit, learn best practices, ask a question or grow your community—please visit our website at www.uidaho.edu/ECHO where you can register to attend, sign-up to receive announcements, donate, and find out more information about our programs.

[Fade out banjo music]

Season three of <u>Something for the Pain</u> is brought to you by ECHO Idaho, supported by the WWAMI Medical Education Program and the University of Idaho, and is made possible with funding provided by BJA, the Bureau of Justice Assistance.

[cue guitar strum and guitar theme w/ lyrics in background]

We here at ECHO also want to hear your feedback. We welcome your questions, comments and suggestions and invite you to email us at <a href="mailto:echoidaho@uidaho.edu">echoidaho@uidaho.edu</a>. And don't forget to subscribe to <a href="mailto:Something for the Pain">Something for the Pain</a> using your podcast app. And if you have a moment, write us a review!

[bring up theme song lyrics and chorus until first "echo Idaho", then drop volume and continue playing]

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The contributing voices on today's episode were those of: [Abby Davids, Elsbeth Jensen-Otsu, Ian Troesoyer, and Winslow Gerrish].

We'd also like to thank all of our listeners, without whom none of this would be possible. Without you, we'd just be talking to ourselves.

[Continue to theme chorus, fade]