Clinical Education Initiative
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HIV OPIOID/ PAIN MANAGEMENT STRATEGIES

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[video transcript]

[00:00:07] All right. My name is Wayne Henderson.

[00:00:09] I'm a Neurologist. I do pain management headache medicine in San Francisco and I wanted to try to keep this one really clinical. Try to keep it more in clinical URL's and that kind of information rather than give a bunch of citations.

[00:00:24] So, hopefully I can accomplish that. So, first off there we go.

[00:00:32] The title of the lecture is the opioid and pain management strategies. And I have no disclosures in regard to anything about this and the objectives are to identify modalities and hopefully actually provide better care. That's the real main idea.

[00:00:49] So neuropathic and nociceptive pain, I'm not going to go through these [inaudible], but the idea is that neuropathic pain and nociceptive pain do have different treatment options completely, so it doesn't matter what type of pain it is.

[00:01:04] So, neuropathic pain is the type of pain that is associated with nerve problems and nerve problems are the ones that are typically found in HIV disease itself.

[00:01:14] The HIV virus is associated with dysfunction of nerves and therefore neuropathic pain is one of the more common types of pain. The HIV related peripheral neuropathy, for example, is a neuropathic pain and then also that toxic neuropathies. I threw in post-herpetic neuralgia here because it's the archetype for the clinical studies. For example, you'll see gabapentin will be FDA approved for post-herpetic neuralgia they use that as the standard for for testing medications. As far as the HIV related neuropathies, the distal sensory neuropathy is one that I think everybody listening to this is quite familiar with and that I think everybody knows it is related to chronic immune activation or, as the studies like to say, so we believe. Now, the nice thing about this one is that it is that it gives an advantage for early treatment if we need to try to convince somebody for a reason aside from the reasons that you already tell people that you want to do early treatment of the condition, this is one good rationale for doing that. It is associated with more advanced disease and therefore early treatment may reduce the incidence. The toxic neuropathy, we don't see that so much anymore as you know because it's related to the old "d drugs", but it is still around and it was related to mitochondrial toxicity when you do see it. Then there's all kinds of other neuropathy that are common. I always tell people

that you can certainly have any normal regular type of neuropathy that any person can get regardless of what underlying likelihood you may have for a specific type.

[00:03:02] And alcohol, nutritional, diabetes, thyroid, they're all common. Although, a good question here is, does anybody know what the most common neuropathy in the world is? And the answer to that one is idiopathic. Then, you know, idiopathic probably many of those will be some very rare type of neuropathy. But, in fact, most neuropathies we never actually find a cause for. Diabetes is actually not number one. So, this is one of those things that I always tell people I try very hard not to speak in medical terms when I'm talking to patients and I try to to come up with all kinds of analogies. And one of the things that I try to explain to people is that peripheral neuropathies are truly peripheral and they're a dying back phenomenon. So, if imagine the nurse coming out of your spinal cord, and I have no idea what I look like on camera, but if you stick your hands out like this and you stick your legs out straight in front of you, the furtherest thing stay away from the spine is your feet. And because that's the furthest thing away, that's where the neuropathy is going to start. And as the neuropathy starts coming up the legs, about the time it hits the knees is about to time the hands get involved. So, people always wonder you know why are my hands not involved in a peripheral neuropathy and that's the reason why, it's just a distance factor and it's actually not any more complex than that. However, there is always an exception to everything.

[00:04:30] And the one exception to that is when people used to have hepatitis C treatment with Pegasys, pegylated interferon, that caused a peripheral neuropathy, oddly, of a temporary basis during treatment and for a few months after, in the arms and the hands and not specifically in the feet. And I've never actually been able to explain that one medically speaking. So, as we move through, nonopioid pain strategies so, neuropathic pain can respond to opioids but it is usually for neuropathic pain considered a last resort. And let's be honest, in today's world opioids are publicly, shall we say, a last resort now for most things because of the news of the opioid epidemic epidemic and all. Even if we may or may not actually call it an epidemic. You know I won't get political on that one but these non-medication modalities are are definitely helpful. Evidence is somewhat lacking with the non medication modalities and so it can be very hard with insurance companies and it can be kind of a push. Cognitive behavioral therapy is perhaps my favorite treatment option for a non medication modality because there is evidence behind it. Unfortunately, as you know, at least yet, there isn't parity as far as payment for it in the health care system but when it's available I find it to be very beneficial. Acupuncture has interesting evidence behind it. The evidence right now is that it's better than placebo but it is no better than sham acupuncture and sham acupuncture and genuine acupuncture have about the same evidence being better than placebo. So, the idea that I told patients was well it's better than placebo and therefore it may be a benefit for you. And everybody should just try what they feel that they are comfortable with. Now, neuropathic medications and and some of the options.

[00:06:28] Everybody knows gabapentin people use gabapentin for pretty much everything in this world right now. And oddly enough the FDA approval for gabapentin, in terms of pain, is only post-herpetic neuralgia it is actually not FDA approved even for fibromyalgia and people assume it is. It has one and only one FDA approval. Pregabalin, on the other hand, has more neuropathic pain indications and tricyclic antidepressants basically have none, and the other ones have variable ones. One of the ones that seems to be coming out right now is Lamotragine, people are beginning to use that for various things. Certainly, I see it a lot and the headache world. It turns out that it's not currently recommended, but we're going to get into that in a second. So, here is the clinical pearl. Whatever happens in the clinic is that people will come in, they'll try gabapentin, they'll try it for one night, "Oh my goodness! it made so dizzy and tired and I stopped it". They come back in two weeks for all up and it's like "I tried gabapentin it failed and now you need to put me on something else. And what I tell people to make sure this doesn't happen is if even if I need to I'll start with pediatric doses essentially, I'll start with 100 milligrams of gabapentin at bad time, if I need to. If that's the medication I'm using and certainly if I'm using a different medication kind of the equivalent junior size dose. And I will gradually build it and I will tell them that, you know, every time you build it that new dose you're getting is new to your body. If you started at 300 at bed time, you know, with gabapentin for example, when you go to six hundred you've just doubled your dose.

[00:08:08] And so your body is going to be tired and sleepy for a few days until your body gets used to it. Then, after you get used to that, if you go to 900, you just increased it 50 percent again. And so you're going to be tired and you're going to all that is going to happen again everytime you go up for a few days. And then I point out that most of these medications can take up to eight weeks before they begin to have clinical benefit. That means that you're going to experience tiredness and dizziness on an on an intermittent basis for two months before you're going to get any benefit out of it. And when people understand that it can take up to two months before it begins to work, then they're not going to give up on it in one week or two weeks and it is going to be an endless cycle of coming back to the clinic and I tried that now try me on pregabalin, now try me on nortriptyline ,now try me or what have you; as it goes on and on. And so, if there's anything that I can comment on neuropathic pain medications, probably the most important thing is that it can take a couple months to begin to work; not to work well, but to begin to work. So, nonopioid pain strategies, to summarize, I'm going to put the I'm almost done looking at the time. First, treat the HIV itself because some of the neuropathic pain that we get is from untreated HIV. Then gabapentin is currently considered a first line in the 2017 guidelines. Capsaicin is a topical medication that is currently with good evidence. Medical cannabis, you know, right now the evidence for all of that is likely to change because, you know, as it becomes legal, as of yesterday, apparently in California here we have waiting lights are on the block for the recreational cannibus. Now that is legal in many places. Hopefully it can be better studied and we're going to be able to learn more about whether it is or is not a treatment for this that or the other.

[00:10:00] So hopefully we'll get more solid evidence one way or the other on that. Right now, oddly enough, the preliminary evidence is that it's not actually that great for pain, interestingly enough. But

that again may change. And then all the other conditions down there right now the current thing is that lamotragine does not have evidence supporting its use. Then, finally second choices, the serotonin or epinephrine uptake inhibitors. You know regular, there is a belief out there that, you know, tricyclists seem to work for neuropathic pain off label, so if they do then the newer antidepressants that are not tricyclists, that are SSRI's, must work too. Well it turns out that the pain benefit of tricyclists is a specific side effect of the tricycling combination or tricyclic drugs, not actually the antidepressant effect. So, the SSRI's do not have the same type of pain killing ability the tricyclists did. So, it's not the idea of I'm just going to put somebody on an antidepressant and because the old ones were for pain the new ones should too. It was a different different concept there. However, the SNRI's do have some painkilling effect. All right, opioid's.

[00:11:18] There's really no slides on the opioids, I want to kind of talk about them in more lay terms. We already know that they can increase pain in the long term. It's what I tell patients here is that as you use opioids what they do is they take the brains that are in the back of the brain, the nerves in the back of the brain in that pain generating center that's back there and they, well, then pain is all in my head. It's not all in my head, well and kind of some other pain generating is in your head, but not in your consciousness so to speak. And these nerves have channels that allow pain transmitting nerves to go through the spinal cord up to your brain, and the more opioids you use the more channels are open for pain to be transmitted. And so, every time you stop your opioid medication it seems like your pain got worse, but that's actually not your baseline pain, that's actually an increased pain, sort of a pain withdrawal, if you will. And the original pain, in some cases, may have even burned out. I have worked on getting people off of opioids in my clinic and of every 50 that end opioids, only about 2 actually ever go back on them. So, a lot of times pain actually gets better. They are still common and standard of care. The special concerns are the cytochrome P450 issues. We all know that. So, I want to talk about the strategies, I don't want to talk about, you know, talking about cytochrome P450, everybody listening is well aware of that. So, what do you do?

[00:12:48] So, I tell people if you're about to change your HIV therapy, let's also go down on your pain management because there are interactions that can make your management suddenly get stronger. I just leave it at that for the clinical aspect of it. And what happens is is, let's just assume somebody is on 100 microgram patch for fentanyl, just because 100 is easy. I tell people as we're changing our therapy we're going to go to 75 and then we're going to go down to 50. And the reason we're going to do this is because as we change your therapy the 50 actually may be a lot stronger than you think it will. Now, there's a little bit of a leap of faith in doing that. But, the idea behind it is usually when I do that, when a patient has to go back up on the fentanyl, we usually don't have to go up past 75. You see what happens is it gives us a chance to start to taper the opioids, not secretly because it's done openly with them, but it gives us that opportunity to do it, because as we go back up their pain benefit does not appear to be necessary to go back up to the 100. Here's what happened when people came to the clinic. Because these medications take time to work, even the opioids do, people would go up and they would keep

titrating to a higher dose and not wait long enough at the current dose. So, when the time comes that they, maybe the 100 patch worked, it was actually the 75 that it would have worked in the first place.

[00:14:14] So, as we go back down a few steps, and go back up, hopefully we don't have to go up more than one step. Another thing that I do is I try to convert everything to Vicodin and equivalence, which would be hydrocodone equivalence. This is the last thing that I actually have to say. Vicodin or hydrocodone is one to one with morphine. That is a one to one conversion. People assume that hydrocodone is weaker than morphine, and patients especially do. So, what I tell people is Let's say you're on 90 milligrams of MS-Contin, 30 Q8, will just say that: extended release morphine. I tell patients well you're on 18 Vicodin a day or 18 hydrocodone a day in equivalent. So, there should be no fear of going from 18 to 17, that should not be that hard to do. And if you're on 17 hydrocodone equivalent a day we could go to 17 to 16. It makes tapering them easier if you're going to taper between the conversions. So, methadone has a special problem associated with it. It has a toxic byproduct and that toxic byproduct has delayed toxicity. One of the most interesting things about methadone is is that you can find a dose that works today, and you can titrate to the dose that works today, but your toxicity may be two weeks from now and you have an overdose two weeks from now. So, sometimes, this is very interesting with methadone, and methadone is unique in this, I could find you the right dose today on Monday, Wednesday, Whatever today is, and two Wednesdays from now you can have an overdose death on the dose that I put you on today. Even though for two weeks you tolerated the medication very well.

[00:15:51] So, there are delayed toxicities with methadone and titration of methadone needs to go exceedingly slowly and in fact I tend not to even use it my own patients at all. Benzodiazepines are never recommended in conjunction with opioids anymore in general. And so, looking at the final slides it's basically just the conclusions of it. So, that's all I have to say. I hope that that was helpful in some way. Any questions?

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