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Three Letters You Need to Know If You Have Fibromyalgia: LDN (/blog/2016/2/28/the-three-letters-you-need-to-know-if-you-have-fibromyalgia-ldn)

By Ginevra Liptan, MD

If you have fibromyalgia there is a treatment option for chronic pain you need to know about. It is called low-dose naltrexone (LDN). Here is the really amazing thing: it is not an opiate pain medication. In fact, it is exactly the opposite: an opiate blocker!

Can we turn down the volume on pain?
(Photo by Andrea Danti/Hemera / Getty Images)

Naltrexone is an opiate-blocking medication that is prescribed in higher doses (50 mg) to treat opiate and alcohol addiction. But when taken at very low doses (1-5 mg dosage range) it reduces pain in two ways. First, it increases the release of endorphins (the body's natural opiates) and second, it lowers inflammation in the central nervous system.

Research has shown that inflammation around the nerves in the central nervous system (brain and spinal cord) is the key factor in the transition from acute pain to chronic pain. And it is very hard to reduce this type of inflammation, so finding something that does it well, and is safe and inexpensive (around $40 per month cash price) like low-dose naltrexone… this could be huge!

How does LDN work for pain? It interacts directly with specific receptors on glial cells in the brain and spinal cord. Glial cells, or glia, are immune and support cells that surround nerve cells and usually remain dormant until triggered for action, like the National Guard of the central nervous system. Glial cells used to be thought of as just inert packing material—in fact, "glia" means "glue"—but we are learning more about the importance of glial cells, especially in chronic pain. They have been called "the new frontier in pain medicine" (Pradeep Chopra, MD, speaker at the LDN2016 conference (http://www.ldn2016.com/)).

The brain can handle a short period of pain signals fairly well, but once the pain signals become chronic this activates the glial cells to start releasing inflammatory chemicals. These inflammatory chemicals irritate the nerve cells around them and cause them to become hypersensitized to pain. Think of the difference between the pain of a papercut, compared to the pain after getting lemon juice in a papercut. The lemon juice hasn't made the papercut bigger, but it has now hypersensitized the nerve cells in the papercut so they cause way more pain. Activated glial cells are essentially spraying lemon juice onto nearby nerve cells, and triggering them to be hypersensitive to pain.

Most of the common fibromyalgia medications like pregabalin (Lyrica) and gabapentin (Neurontin) aim to quiet the pain nerve signals themselves. But these treatments are only partly or temporarily effective, because the nerves are still getting barraged by the "lemon juice" of inflammation. A much better way to reduce pain is to calm the glial cells back into going dormant, and that is exactly what LDN does. LDN acts on specific receptors on the glial cells that essentially tell them to go back into hibernation and stop releasing inflammatory chemicals. This allows the nerve cells to normalize the volume on pain signals again. LDN's ability to reduce central nervous system inflammation means it has huge potential for treating autoimmune diseases, including multiple sclerosis and lupus.

I have had quite a few fibromyalgia patients get impressive pain reduction with low dose

naltrexone. In two studies done at Stanford University, LDN reduced fibromyalgia pain, with 57% of the participants reporting a significant (1/3) reduction of pain. They reported that LDN was generally well tolerated and that side effects were infrequent, but included headache, vivid dreams, insomnia, or anxiety, and that matches my clinical experience as well.

However, there are two current challenges to prescribing LDN. First, it has to be specially made by a compounding pharmacy and many doctors are not used to writing compounded prescriptions. Second, low dose naltrexone potentially does not mix well with opiate pain medications because it might induce withdrawal symptoms or counteract the pain relief of opiates (although this may not be as much of a concern as we thought; stay tuned for my next post where I explore some new evidence that it may be actually be beneficial to add LDN to opiates). For now, I advise patients on long-acting opiates to avoid LDN and those that take short-acting pain meds to take them at least six hours apart from LDN to avoid any possible negative interactions.

Unfortunately, since naltrexone is a generic medication there is no pharmaceutical industry interest in funding studies, so the research has primarily been a grassroots efforts thanks to organizations like LDN Research Trust (http://www.ldnresearchtrust.org/), who just hosted a very interesting conference that I was able to attend via live stream.

To learn more about LDN, check out *The LDN Book* (http://amzn.to/1T3B3Bc), just released this month.

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