Science to patients (Wetenschap voor Patiënten)
Webinar 70: Neuroinflammation and ME/cfs

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Have neuroimflammations been shown in ME/cfs?

The question of neuroinflammation is interesting because there's been some conflict between the United States criteria for CFS and other criteria using ME because of the term encephalomyelitis. Because technically 'itis' means inflammation and we didn't really have strong evidence of neuroinflammation earlier. But in the last year or two there have been very promising studies published showing the presence of neuroinflammation--, PET scan, MRI scans. They are small but high-quality studiesy. They still need to be replicated but I think that they are providing evidence that neuroinflammation is present in ME.

Which part of the nervous system is affected as usual?

Knowing which part of the nervous system is affected is a challenge when we lack the objective markers. But the study showing there are studies showing neuroinflammation point to white matter disease as for the site of neuroinflammation. We do know though that our patients may have involvement of the peripheral nervous system in terms of auto antibodies. So, the interplay of the peripheral and central nervous system make a complex cycle that may be difficult to tease apart.

Neuroimflammations in ME/cfs: cause or consequence of other symptoms?

Knowing if neuroinflammation is the cause or the consequence of ME is going to be a challenge. I'm not sure we understand that at this point. We do think there may be a genetic predisposition or infectious triggers or viral triggers, mechanical injury, exposures and other kinds of factors that lead to neuroinflammation. So it doesn't arise probably from nothing. As I said earlier it's also possible that the peripheral nervous system becomes dysfunctional, even the example of small fiber polyneuropathy. If those damaged nerves out at the very periphery of the skin are sending abnormal signals to the brain it may set up a sequence of changes in the brain that create change. So we have many many questions to answer about the interplay of the peripheral and central nervous system along with autoimmunity in the periphery and neuroimmune mechanisms in the brain.

Are there treatments for neuroinflammation in ME/cfs?

We don't really have treatments for neuroinflammation in ME at this point. Particularly not treatments that are proven or tested. We may be able to borrow medications from other conditions of neuroimmune mechanisms, but that remains to be seen. There is one interesting treatment that has some published studies and that's the use of naltrexone, a very low dose naltrexone, which might be modulating glial-cell inflammation in a way that is treating at least a part of neuroinflammation. Maybe the part more related to pain amplification.

What effect do antivirals have?

There's been some interest in the use of antivirals in ME/cfs. The relationship of with this to neuroinflammation is not immediately clear. If there is persistent or reactivating herpes family virus infection then the use of antivirals may be very effective to prevent that or to really decrease the activity of the virus which might be triggering continuing neuroinflammation. But there are many patients with ME that don't respond to antivirals. And the response to antivirals is exceedingly slow in most cases and many patients relapse when the antiviral medications are stopped. So while we are interested in trying to understand why it works and to use any treatment that might be helpful for people at this point, it has limited benefit. And many of the antivirals are very expensive and bring some toxicities.

So I think it's important that we understand what we're treating and get the clinical trials done to know in what situation antivirals would play the most important role. Drugs like immune modulators, like rituximab, work in a different way directly on immune cells that are either playing a role themselves or are producing antibodies, autoantibodies that may be playing a role. If patients get better with an immune modulator but not an anti-viral then how do we live with the idea that there's a chronic activated viral infection that is driving the illness. These are very important questions to pursue. It may have something to do with stage of illness. That early stage illness is really different from medium and from late-stage illness.