

## Autonomic dysfunction, small fiber neuropathy and Lyme disease

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The [retrospective study included 10 patients](#) diagnosed with post-treatment Lyme disease syndrome, who had autonomic testing performed between 2016 and 2018 at the Brigham and Women's Faulkner Hospital Autonomic laboratory. [1]

The authors aimed to identify SFN as a possible biomarker of "PTLDS," in addition to evaluating autonomic dysfunction associated with presumed small fiber neuropathy and assessing cerebral blood flow, since cognitive complaints may be due to cerebral hypoperfusion.

Novak and colleagues defined "PTLDS" using the Aucott's criteria, which states that patients must have:

1. A combination of fatigue, cognitive complaints and chronic widespread pain following the treatment of Lyme disease for at least a 6-months period;
2. An absence of other disorders that can explain the complaints associated with "PTLDS";
3. A documented history of Lyme disease satisfying the CDC criteria.

Their 10 patients presented with a broad range of symptoms. The most common included lightheadedness (n=8), dry mouth or dry eyes (n=8), pale or blue feet (n=6), feet colder than the rest of the body (n=6), decreased sweating at feet at rest (n=5), and decreased sweating at feet after exercise or during hot weather (n=5).

Symptoms reported by study participants. (Click on image to enlarge.)

Pain was frequently reported and described as: aching pain (n=10), numbness (n=8), prickling sensation (n=8), burning pain (8), lancinating pain (6), and allodynia (n=6). (Allodynia refers to central pain sensitization following a normally non-painful stimulation.)

Less common symptoms were: sweating increased at hands (n=3), nausea, vomiting, or bloating after meal (n=2), persistent diarrhea (n=4), leaking of urine (n=3), persistent constipation (n=2), and difficulty in erection (n=1).

All of the subjects had previously undergone routine autonomic testing. Novak and colleagues performed skin biopsies from the right calf using a 3-mm circular disposable punch tool.

They found that more than 50% (7 out of 10) of patients had a low cerebral blood flow velocity (CBFv) from their middle cerebral artery. This was determined using a Transcranial Doppler. The study was not designed to determine if the low CBFv contributed to the cognitive impairment in those 7 patients.

All of the patients had a loss of small fibers, along with autonomic dysfunction and abnormal cerebral blood flow.

According to Novak, the sensory symptoms and pain reported by their patients may have been caused by small fiber neuropathy and low cerebral blood volume. “SFN appears to be associated with 'PTLDS' and may be responsible for certain sensory symptoms,” the authors write. “Dysautonomia related to SFN and abnormal CBFv also seem to be linked to 'PTLDS'.”

Therefore, “Our study indicates that SFN may be an objective marker of 'PTLDS', at least in patients with prominent sensory symptoms,” Novak writes.

The study was not designed to address treatment. The authors assumed the patients did not have a persistent infection.

Editor’s note: I have been reluctant to use the term “PTLDS” until there is a reliable test to rule out a persistent infection.

**Related Articles:**

[Can we measure the brain's exaggerated response to pain and sensory input?](#)

[Is your fibromyalgia patient a candidate for Lyme disease treatment?](#)

[Could autonomic dysfunction lead to pain in Lyme disease?](#)

**References:**

1. Novak P, Felsenstein D, Mao C, Octavien NR, Zubcevik N (2019) Association of small fiber neuropathy and post treatment Lyme disease syndrome. PLoS ONE 14(2): e0212222. <https://doi.org/10.1371/journal.pone.0212222>

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