One of America’s top neurologists says that the underlying pathogenesis of neuropathy in type 2 diabetes (T2D) is likely the metabolic syndrome, rather than solely hyperglycemia.

As such, consuming healthy foods and increasing exercise may be the answer to preventing neuropathy brought on by the metabolically induced changes in peripheral nerves of patients with type 2 diabetes, said Eva Feldman, MD, PhD, Professor of Neurology at the University of Michigan in Ann Arbor, Michigan. “We know that in type 1 diabetes, there is a clear decrease in the incidence and prevalence of diabetic neuropathy with good glucose control. But in patients with type 2 diabetes, controlling glucose has very little effect on neuropathy. “And what we saw is that the more components of the metabolic syndrome a patient has, the more likely they are to have neuropathy. “This told us that we have to move away from the glucose-centric idea that neuropathy in type 2 diabetes is just caused by hyperglycemia,” Professor Feldman said.

In Australia, approximately half of all patients with T2D are thought to have diabetic neuropathy. “We know that neuropathy is the leading predictor of mortality in a patient with diabetes, and that it leads to poor quality of life, pain, depression, and ulcers and amputation. “In the US alone, diabetic neuropathy costs the US government about $60 billion annually in healthcare costs,” Professor Feldman said.

Metabolic reprogramming of nerve bioenergetics

In a simple clinical trial conducted at the University of Michigan, Professor Feldman and her team recruited patients in the early stages of T2D and placed them on a diet and exercise intervention. Almost all of the patients were obese and had the metabolic syndrome, and experienced discomfort in their feet.

The primary aim of the study was to assess the effects of diet and exercise on the small unmyelinated nerve fibers that are important for sensation and pain. Each patient was assigned his or her own exercise physiologist and dietician during the first year of follow-up. At the end of year 1, patients who were compliant with the intervention loss 7 kg of their body weight and had a clear increase in the number of intraepidermal nerve fibers. However, when these patients were followed for an additional 2 years, during which they did not have access to the physiologist or dietician, they gained back the weight lost. “When we re-biopsied their skin, we saw that with the return of pain in their feet they had also lost the intraepidermal nerve fiber density gained” at the end of year 1. “So this was a nice clinical proof-of-concept that showed us the importance of controlling aspects of the metabolic syndrome,” Professor Feldman said.

“The underlying pathogenesis of type 2 diabetes is not simply glucose, it’s the metabolic syndrome. And it’s likely that there’s metabolic reprogramming of nerve bioenergetics gone awry – there is energy failure, especially at the distal nerve. “We really need future studies to target these new mechanisms in our patients with early intervention,” she added.

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