Dr. Gurpreet Singh Padda, MD: Diabetic Neuropathy Is Treated as One Nerve Problem; It Is Three Different Problems

For World Diabetes Day, Dr. Padda on the distinct mechanisms behind one diagnostic label, and why the distinction changes what a patient should expect

Regenerve Logo

ST. LOUIS, MO, UNITED STATES, November 14, 2026 / EINPresswire.com/ -- Dr. Gurpreet Singh Padda, MD, MBA, MHP, who directs Regenerve in St. Louis, is using World Diabetes Day to argue that one of the most common complications in medicine is routinely misnamed. Pooled estimates put the share of people with diabetes who develop peripheral neuropathy at roughly 40 percent, with prevalence rising sharply with age and disease duration. As many as half of cases produce no symptoms at all (ADA Standards of Care in Diabetes 2026, section 12, Diabetes Care 2026;49(Suppl. 1):S261).

Of those who do have symptoms, most receive the same explanation, nerve damage from high blood sugar, and a similar short list of medications aimed at muting it. The single label, Dr. Padda argues, conceals at least three mechanisms, which respond to different things and carry different prognoses.

Microvascular starvation. Peripheral nerves depend on the vasa nervorum, the fine capillary network supplying them. When those vessels are compromised the nerve is ischemic before it is structurally damaged. This is a perfusion failure presenting as a sensory one, and it is why some patients describe symptoms that vary with position, temperature or activity. It is also the mechanism with the most room left in it, because a hungry nerve is not yet a destroyed one.

Glycation. Sustained hyperglycemia drives non-enzymatic glycation, producing advanced glycation end products that bind the RAGE receptor and sustain inflammatory and oxidative signaling. Once formed, cross-linked proteins are slow to turn over. The process therefore continues largely independently of current glucose readings, which is why patients whose control has improved often report that their symptoms have not.

Autonomic and structural decay. A third mechanism runs through the polyol pathway. When

glucose is abundant, aldose reductase converts it to sorbitol, consuming NADPH in the process and depleting the glutathione the nerve needs to manage oxidative stress. The result is demyelination and autonomic involvement: loss of protective sensation, altered sweating and temperature regulation, instability in balance and gait.

"Burning feet is a symptom, not a diagnosis," Dr. Padda said. "Three patients can present identically and be suffering three unrelated failures. Treat them the same way and you will help one of them by accident."

Why the distinction is practical

Evaluation begins with electrodiagnostic testing performed on site, nerve conduction studies and needle electromyography, to establish whether a nerve is compressed, metabolically injured, or both, and to localize it. A compressed nerve and a glycated one are not the same clinical problem, and the difference is not reliably apparent from symptoms. A patient describing burning in both feet may have an entrapment that is correctable, a perfusion deficit that is modifiable, or fifteen years of accumulated cross-linking that is not, and the three conversations that follow are nothing alike.

The distinction also sets expectations honestly, which Dr. Padda treats as the more important half of the exercise. A perfusion problem and an entrapment have different ceilings from an accumulated glycation burden, and telling a patient which one they have is more useful than telling them their sugars need to improve. The second statement is usually true and almost never actionable in the way the patient hears it.

"An ischemic nerve and a glycated nerve feel identical to the patient and are nothing alike to the clinician," Dr. Padda said.

Treatment is directed at the mechanism identified: peripheral or vagal nerve stimulation, endoscopic decompression where entrapment is present, photobiomodulation, infusion therapy addressing metabolic and oxidative drivers, alongside nutritional and metabolic care. Balance and gait training, durable medical equipment and wound care address the mechanical consequences of lost protective sensation, which arrive whether or not the patient ever had pain.

The legacy effect

The hardest conversation in the clinic, Dr. Padda says, is with the patient who has done exactly what was asked of them.

Glycation is cumulative and slow to reverse. A person who spent fifteen years with poor control and the last two with excellent control is carrying the chemistry of the first fifteen in tissues that turn over on a timescale of years, not months. Their A1c reports the last three months. Their

nerves are reporting on a much longer record.

That is not an argument against control. Control going forward is necessary, and it changes what accumulates from here. It is an argument against promising that improved numbers will retroactively undo a burden that was laid down before those numbers existed, and against letting a patient conclude they have failed when the biology is simply slower than the lab.

"The A1c tells you about the last three months," Dr. Padda said. "The nerve is reporting on the last fifteen years."

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How glucose damages the nervous system: https://regenerve.com/glycation-how-sugar-damages-nerves/
When burning feet are compression rather than neuropathy: https://regenerve.com/nerve-compression-carpal-tunnel-sciatica-double-crush/
Reversing Diabetes MD, why diabetic nerve pain starts before the diagnosis: https://reversingdiabetesmd.com/diabetic-nerve-pain-metabolic-cause/

About Regenerve

Regenerve, Advanced Peripheral Neuropathy Treatment Center, is a physician-led clinic in St. Louis, minutes from St. Louis Lambert International Airport, directed by Dr. Gurpreet Singh Padda, MD, MBA, MHP, a board-certified pain physician. Care targets the underlying causes of nerve damage: metabolic, autoimmune, nutritional, structural and toxic. Regenerve is a DBA of Interventional Center for Pain Management, P.C., and serves Missouri and Illinois.

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