COMMENT ON JAISWAL ET AL.

Prevalence of and Risk Factors for Diabetic Peripheral Neuropathy in Youth With Type 1 and Type 2 Diabetes: SEARCH for Diabetes in Youth Study. Diabetes Care 2017;40:1226–1232

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We were interested by the recent article by Jaiswal et al. (1), who reported a high prevalence (22%) of diabetic peripheral neuropathy (DPN) in young subjects with type 2 diabetes in the SEARCH for Diabetes in Youth (SEARCH) study. Although significance was not reached for all the analyses in the type 2 diabetes group, long-term poor glycemic control, dyslipidemia, and smoking emerged as modifiable risk factors, and the authors suggested that their management may help to prevent or delay irreversible nerve damage. This optimistic view is, however, challenged by the results of multifactorial interventions: no improvement of vibration perception thresholds (VPT) in the Steno-2 study (2) and only a small initial improvement with later seemingly inexorable progression of Michigan Neuropathy Screening Instrument (MNSI) scores in the Look AHEAD (Action for Health in Diabetes) trial (3). At the present time, established DPN seems to be irreversible, as underlined by Coppini et al. (4).

This may not be the case, however, in patients with early forms of DPN. The Toronto Consensus Panel defined subclinical DPN by the presence of electrophysiological abnormalities in patients who are free of clinical signs and symptoms of DPN (5). Identifiyng these patients is difficult because it requires a time-consuming electrophysiological study, but the use of point-of-care electromyography (DPNCheck; NeuroMetrix) may help. Is subclinical DPN frequent? Can we predict it from more simple measurements?

In patients without evidence of symptomatic neuropathy (no neuropathic pain, ankle reflexes present, normal monofilament and turning fork testings), we measured nerve conduction velocity and amplitude potential with a DPNCheck to identify subclinical neuropathy. Then, we measured their muscular strength by handgrip, electrochemical sweat conductance by Sudoscan (Impeto Medical), and VPT by neuroesthesiometry to detect a subclinical functional impairment.

The 26 patients (15 men) were aged 60 $\pm$ 10 years. Their diabetes duration was 13 $\pm$ 8 years and their HbA_{1c} 8.8 $\pm$ 1.9%. Eight had a subclinical neuropathy (nerve conduction velocity $<$40 m/s and/or amplitude potential $<$4 μV with DPNCheck). We compared them with those without neuropathy. They did not differ in diabetes duration, BMI, or associated retinopathy or nephropathy. There was no difference in electrochemical sweat conductance or muscular strength. The median VPT was increased in patients with subclinical neuropathy compared with people without neuropathy (11.5 [5.5–17.8] vs.7.0 [4.8–9.0] V, respectively; $P < 0.05$).

A noticeable proportion (30%) of our patients without clinical signs had subclinical DPN, and their slightly elevated VPT could help to identify them, although the values were much lower than the levels known to predict the later occurrence of foot ulcers (25 V). At such levels, Coppini et al. (4) have shown that well-controlled glucose and lipid levels prevent the natural increase of VPT over 7 years of follow-up, which suggests that early interventions can be beneficial. Although Jaiswal et al. (1) did not mention any electrophysiological study, it seems probable that an important proportion of the young patients included in the SEARCH study had subclinical DPN. As the MNSI score includes a graduated VPT estimation (absent = 1, reduced = 0.5, present = 0), it would be interesting to know the proportion of intermediate (0.5) VPT and whether this presumably early stage of DPN was related to modifiable risk factors.

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