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Letter to the Editor

Diagnosing Small Fibre Neuropathy is based on Clearly Defined Criteria, which Require Sequential Application of Various Tools

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The article by Jänsch *et al*. is excellent but some points should be discussed [2].
The first point is the design of the study. Retrospective designs have the disadvantage of not being able to control the accuracy of the stored data, not systematically applying the same examinations to all patients, producing missing data, and not being able to generate and add desirable new data to the data set. Particularly in the case of pain, hypoesthesias, dysesthesias, or paresthesias, as well as autonomic disturbances, it is often necessary to question the patient about the accuracy of the previous information. Sensory disturbances may be transient in nature, fluctuating, and often migratory and may exhibit dynamic changes in distribution, intensity, quality, and persistence.
A second point is that SFN may affect not only nociceptors in the skin, but also other dermal receptors or autonomic fibres that innervate arterioles, sweat glands, hair follicles, or sebaceous glands. In addition, SFN may also be due to affection of spinal ganglion cells leading to regional pain or sensory syndromes. Therefore, the clinical presentation of SFN is not only limited to acral pain and sensory disturbances but may also include autonomic dysfunction.
A third point is that the diagnosis of SFN is based not only on the individual and family history, clinical presentation, and PGB 9.5 stained skin biopsy, but also on several other instrumental examinations. These include quantitative sensory testing, sensory stimulation testing, microneurography of C-fibres of the superficial peroneal nerve, autonomic testing (e.g. deep breathing, Valsalva maneuver, tilt test, cerebral blood flow measurements, quantitative sudomotor axon reflex), and corneal confocal microscopy, pain-evoked potentials, laser speckle contact analysis, laser Doppler flowmetry, contact heat-induced potentials, and skin biopsy assessing sweat gland nerve fibre density or the number of peptidic fibers [1]. Autonomic dysfunction can also be assessed by applying Fourier analysis to long-term ECG recordings. These methods should be used sequentially when SFN is suspected but previous investigations were inconclusive. If IENFD is normal, SGNFD may be abnormal.
A fourth point is that SFN is not only characterised by acral pain, as mentioned in the introduction [2]. Pain in SFN can have a very variable distribution. It can be focal, regional, or global. For example, SFN can also manifest as burning mouth syndrome or restless genital syndrome [1].
A fifth point is that combinations of pain characters, pain locations, pain triggers, and pain-relieving factors were not analysed. Since clinical experience shows that there is not just one type of pain, one location of pain, one trigger, or one alleviating factor in a patient, it would be desirable to know how many patients combinations of these factors.
A sixth point is the discrepancy between the mean time from symptom onset to diagnosis, reported in table 1 for SFN patients as 2.8 years, while the difference between mean age at symptom onset and mean age at diagnosis was 6 years [2]. This discrepancy should be resolved.
In conclusion, the diagnosis of SFN is based on well-defined clinical criteria that require the sequential application of various sophisticated tools until all differentials are excluded.

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