



A study to Assess the Neuropathy in Subclinical Nerve Lesions among Patients With Type 2 Diabetes patients at selected Hospitals in Indore

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Introduction

There is a clear link between the existence of distal symmetric diabetic polyneuropathy and an increased risk of death in diabetics. Tingling, sudden pain or burning sensations, hyperalgesia, allodynia, or extreme sensitivity to temperature changes are all indications of DPN. There are several anatomical and functional variations in the central nervous system between persons with and without painful diabetic neuropathy. Nevertheless, no differentiating markers of the peripheral nervous system have been established to date to differentiate between painful and painless diabetic neuropathy. Additionally, the origins and evolution of DPN remain unknown. Both prediabetes and diabetes-related nerve damage are considered to be caused by the loss of microscopically tiny, unmyelinated C fibres. In hindsight, the sickness seems to have developed by a process of demyelination and axonal degeneration of myelinated fibres. Because to the difficulties and hazards of acquiring nerve tissue, research into the underlying pathophysiological components of DPN in people is restricted. Moreover, since nerve biopsies may only be conducted on distal nerves, proximal fibres cannot be assessed. Developing noninvasive, objective, in vivo techniques for identifying and accurately localising DPN nerve damage at an early stage is thus critical for understanding the pathogenesis and assessing prospective therapeutic choices. Recent research has shown that 3-Tesla magnetic resonance imaging of the brain provides substantially more information than 2-Tesla imaging. With the use of Tesla, DPN peripheral nerve lesions may be diagnosed and properly localised. It has been shown that hyperintense nerve lesions in a T2-weighted, fat-suppressed imaging sequence are related with poor nerve conduction qualities. Yet, the specific clinical impact and link to nerve fibre types of lesions discovered by MRN have not been properly investigated, since lesions have so far only been related with basic clinical evaluations and electrophysiological tests. As a consequence, it's unclear which types of nerve fibres are affected by T2w lesions. Moreover, detecting early stages of diabetic neuropathy may be useful for future clinical trials assessing different treatment options. The most sensitive clinical strategy for characterization of neuronal injury is complete quantitative sensory testing, which includes mechanical testing for large-fiber function and has yet to be used to determine the clinical importance of MRN. As a result, we conducted a case control study in patients with type 2 diabetes, both with and without DPN, as well as in healthy controls, using MRN, EPT, and QST as the potentially most sensitive and specific noninvasive clinical methods for evaluating both the exact clinical and neurophysiological status, as well as the load of structural nerve lesions.

Methodology

Individuals with type 2 diabetes, both with and without DPN, were included, as were patients without diabetes and neuropathy. For this investigation, a case control approach was used. To diagnose DPN, all participants were questioned about DPN-related symptoms in order to calculate the Neuropathy Symptom Score. The research excluded patients with persistent alcoholism, end-stage renal illness, Parkinson disease, rheumatic autoimmune disorders, malignant tumours, or spinal abnormalities. A fasting blood sample was conducted to assess HbA1c, creatinine, and serum lipids, as well as the urine albumin/creatinine ratio.

Results

There were a total of 100 participants: 50 people with type 2 diabetes and an NDS of 0; 50 people without diabetes and an NSS and NDS of 0; and 100 people with type 2 diabetes and an NDS of 3. MRN was performed on each and every topic. The hyperintense lesion burden that was determined using T2w spanned the range of 70% to 100%. In the MRN study, there was no relationship found between clinical complaints and the amount of T2w hyperintense lesions. On the other hand, tingling was shown to have a significant correlation with both MDT ($r = 0.33$) and VDT ($r = 0.217$). Male sex was associated with a greater T2w hyperintense lesion load ($r = 0.62$), and this association remained even when only patients with diabetes and DPN were included in the analysis ($r = 0.208$). This was determined through a regression analysis that included all of the participants in the study. Diabetes duration was another predictor of lesion load ($r = 0.44$) among diabetics who had diabetic peripheral neuropathy (DPN). Lesion burden was not linked with age, HbA1c, BMI, uACR, insulin usage, alcohol use, smoking, coronary heart disease, or peripheral artery disease in any of the trials.

Conclusion

This study adds to the growing body of evidence suggesting that lesions in T2w MRN are a normal finding in peripheral nerves associated with the ageing process. Decreased activity in the nerve's medium and large fibres is most often associated with these conditions. Given the association between sciatic nerve lesion load and diabetes duration and sensory and motor nerve function in patients with diabetic peripheral neuropathy and normal persons, it is likely that lesions play a pathophysiological role in DPN. Hence, future studies should use a cohort strategy to investigate the causes and effects of these lesions in both diabetic and non-diabetic populations.

REFERENCE

- 1) Bertora, P., Valla, P., Dezuanni, E. et al. Prevalence of subclinical neuropathy in diabetic patients: assessment by study of conduction velocity distribution within motor and sensory nerve fibres. *J Neurol* 245, 81–86 (1998). <https://doi.org/10.1007/s004150050182>
- 2) Hasani N, Khosrawi S, Hashemipour M, et al.: Prevalence and related risk-factors of peripheral neuropathy in children with insulin-dependent diabetes mellitus. *J Res Med Sci*. 2013, 18:132-6
- 3) Hajas G, Kissova V, Tirpakova A: A 10-yr follow-up study for the detection of peripheral neuropathy in young patients with type 1 diabetes. *Pediatr Diabetes*. 2016, 17:632-41. [10.1111/pedi.12382](https://doi.org/10.1111/pedi.12382)
- 4) Hyllienmark L, Alstrand N, Jonsson B, Ludvigsson J, Cooray G, Wahlberg-Topp J: Early electrophysiological abnormalities and clinical neuropathy: a prospective study in patients with type 1 diabetes. *Diabetes Care*. 2013, 36:3187-94. [10.2337/dc12-2226](https://doi.org/10.2337/dc12-2226)
- 5) Misra UK, Kalita J, Nair PP: Diagnostic approach to peripheral neuropathy. *Ann Indian Acad Neurol*. 2008, 11:89-97. [10.4103/0972-2327.41875](https://doi.org/10.4103/0972-2327.41875)
- 6) Karsidag S, Morali S, Sargin M, Salman S, Karsidag K, Us O: The electrophysiological findings of subclinical neuropathy in patients with recently diagnosed type 1 diabetes mellitus. *Diabetes Res Clin Pract*. 2005, 67:211-9. [10.1016/j.diabres.2004.07.017](https://doi.org/10.1016/j.diabres.2004.07.017)
- 7) Olsen BS, Johannesen J, Sjølie AK, et al.: Metabolic control and prevalence of microvascular complications in young Danish patients with Type 1 diabetes mellitus. Danish Study Group of Diabetes in Childhood. *Diabet Med*. 1999, 16:79-85. [10.1046/j.1464-5491.1999.00024.x](https://doi.org/10.1046/j.1464-5491.1999.00024.x)
- 8) Ryan CS, Conlee EM, Sharma R, Sorenson EJ, Boon AJ, Laughlin RS: Nerve conduction normal values for electrodiagnosis in pediatric patients. *Muscle Nerve*. 2019, 60:155-60. [10.1002/mus.26499](https://doi.org/10.1002/mus.26499)