

# INDIAN JOURNAL OF MEDICAL SCIENCES

VOLUME	63
NUMBER	4
APRIL	2009

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Published by

**Medknow Publications & Media Pvt. Ltd.**

A-109, Kanara Business Centre, Off Link Rd, Ghatkopar (E), Mumbai 400075, India

# Indian Journal of Medical Sciences

(INCORPORATING THE MEDICAL BULLETIN)

VOLUME 63

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## EDITORIAL

### REVERSIBILITY OF NEURONAL DAMAGE IN DIABETES: THE SEARCH FOR A NEWER THERAPEUTIC PARADIGM

NIHAL THOMAS

Both microvascular and macrovascular complications account for a large burden on the patient with diabetes, and the decline in the quality of life and financial losses incurred are significant for both the patient and the community.<sup>[1]</sup> Multicentric studies in the outpatient departments of large tertiary care referral centers in developing countries have shown that peripheral neuropathy with unimodular diagnostic techniques such as the 2- and 10-grammes monofilaments accounts for nearly 15% of subjects in an outpatient setting.<sup>[2]</sup>

Once diabetic neuropathy has occurred, it may be clinically stratified into symptomatic and nonsymptomatic disease.<sup>[3]</sup> Symptomatic neuropathy may be either associated with positive or negative symptoms. Regression of these symptoms may occur with either good glycemic control or definitive therapeutic maneuvers to mask positive symptoms.<sup>[4]</sup>

The article by Norlinah *et al.*<sup>[5]</sup> in this issue of the journal has shown that improved glycemic control is associated with improved neuronal function as assessed with peroneal motor conduction velocity. This improvement appears to be related to insulin therapy. Though on analysis there is an improvement in neuronal function in a relatively short period of time, the results should be interpreted with caution from a clinical perspective. From a statistical viewpoint, the analysis has been done through simple correlations rather than multiple logistic regression analysis. Therefore, other variables that may be responsible for this improvement may not have been accounted for, which could at times transform the results into something far less significant. The study being single-blinded, which was of course unavoidable, may have resulted in a bias that could at times influence dose adjustment of insulin in these subjects and could have tilted them towards marginally better glycemic control in the insulin arm; therefore, it would be interesting to see if the same results could be replicated with a larger sample size of subjects (thereby obliterating the phenomenon of 'beginner's luck').

The authors have mentioned in their conclusions

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that it would be judicious to screen all patients with diabetes for neuropathy with nerve conduction velocity studies and that those with subclinical neuropathy would benefit from more aggressive therapy. This would not be a practical or a cost-effective strategy to handle the problem. Moreover, nerve conduction anomalies of the peroneal nerve are extremely common in subjects with diabetes who may even have reasonably good control.<sup>[6]</sup> Thus, to base therapeutic decisions on this modality of investigation would be a little farfetched.

However, despite these limitations, the study by Norlinah *et al.* is an important scientific echo of a landmark study published in the 1990s by Partanen *et al.* In a 10-year follow-up, they found that there was a higher incidence of polyneuropathy in those with poorer glycemic control. What was striking was the finding that more profound anomalies in conduction velocity were associated with hypoinsulinemia, a discovery which was independent of the severity of hyperglycemia.<sup>[7]</sup> To dichotomize and differentiate between the impact of the insulin peptide and hyperglycemia may be difficult. However, one could state that the proof would lie in using a peptide of similar configuration or origin to improve neuropathy. Ekberg *et al.* utilized injectable C-peptide for a period of 6 months and were able to demonstrate improvement in conduction velocity and improvement in symptom score.<sup>[8]</sup>

In general, however, prevention is undoubtedly not just better than cure but far superior to cure since reversibility of neuronal damage remains an electrophysiological phenomenon rather than a clinical reality with most forms of treatment. Thus, the quest for a suitable therapeutic agent

for reversal of neuronal damage must still go on.

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DOI: 10.4103/0019-5359.50761

Source of Support: Nil, Conflict of Interest: None declared.