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THESES

A CLINICAL AND NEUROPHYSIOLOGIC RANDOMIZED TRIAL ASSESSING TWO DIFFERENT REGIMENS OF ORAL STEROID TREATMENT OF ULNAR NEUROPATHY IN TYPE 1 AND TYPE 2 LEPROSY REACTIONS (ABSTRACT)*. **THESIS, SÃO PAULO, 2006.**

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Hansen disease's neuropathy impairs during the reactions and frequently develops, with axonal loss, physical deficiencies. The actions regarding the prevention of these incapacities rely on knowing the physiopathologic mechanisms and its treatment. Therefore, it is interesting to analyze the distinct oral steroids regimens and, secondarily, to distinguish the neurophysiologic behaviour of the nerves on type1 leprosy reaction (T1R) and type 2 leprosy reaction (T2R). This experiment was a six-month random clinical and neurophysiologic trial, using the ulnar nerve as a model in Hansen disease's patients, for T1R and T2R, which were sent to Lauro de Souza Lima Institute in Bauru, Brazil. From 163 leprosy patients assessed, 21 patients were selected, 12 with T1R and nine with T2R (42 nerves). Eight nerves did not show signs of being compromised, resulting in a total of 34 nerves with neuropathies. The clinical assessment included the following tests: Graded Sensory Testing (GST) with the Semmes- Weinstein monofilaments; Nerve palpation (NP); Assessment of spontaneous pain with a visual analogical scale (VAS) and Voluntary Muscle Testing (VMT). A final clinical score (CS) was calculated by summation of numerical results for VAE, NP, GST and VMT.

The electrophysiological evaluation consisted on the motor nerve conduction of the ulnar nerve in segments of the wrist, forearm, across the elbow and also in the whole ulnar nerve. The applied parameters were the compound motor action potential (CMAP) elicited on those three sites, the distal latency, the conduction velocity along the forearm and across the elbow, the temporal CMAP dispersion in the elbow, and above it, and the F wave. The patients had been submitted to eight evaluations during this trial's length. Statistical studies verified the significance level among the oscillations noted for each variable in three different times: initial and first week, initial and first month, initial and sixth month. The tests were applied on the results variation compared, each pair, i.e. initial and first week, between each regimen and between each type of reaction.

Prednisone (available in public medical centers) was applied. Two treatment regimens were used for each type of reaction. An experimental group with initial 2 mg/kg/day dosage in the first three days, establishing a 1, 76 mg/kg/day dosage in the first week and a control group with a dosage of 1 mg/kg/day at the same period till the third week. After the third week, on T1R patients, the doses were 1 mg/kg/day in both groups, the reduction was monthly and on T2R patients, the reduction was every 2 days, until 0,5 mg/kg/day dosage levels were reached at the end of the first month. The steroids regimens with higher initial doses showed statistical significant differences either with the T1R or the T2R nerves during the first month. When the final results were compared, there were no significant differences in the period where the doses were similar. When treatment had been established in less than three months from the beginning of the symptoms, there were no relevant differences on the results relating the steroids regimens. The axonal and demyelinating neurophysiologic changes were found along the entire nerve, being more conspicuous across the elbow, in T1R and T2R nerves. The demyelinating events were predominant in T1R when compared to T2R, the same behavior was observed on remyelination under treatment. Thus, the predominance of the myelinic involvement in T1R when compared to T2R, had been evidenced.

The steroids responses were dose dependent in both reactions. Nevertheless, when the treatment had been established early in the process, the responses to the distinct steroids regimens were equivalent. With the neurophysiologic method, it was possible to demonstrate, via statistical analyses, the differences between the distinct steroids regimens and among the ulnar neuropathy behavior in T1R and in T2R. Although the clinical methods had shown gradual response improvement for each regimen's treatment, and for each type of reaction, separately, there were no relevant statistical differences when those tests were submitted to the same kind of comparison made with the neurophysiologic parameters.

KEY WORDS: Hansen's disease, reactions, ulnar neuropathy, steroids, neurophysiology.

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