HANSEN'S DISEASE NEUROPATHY, CLINICAL AND DIAGNOSTIC APPROACH

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In Hansen's Disease (HD) the following aspects are essential in its definitions, classifications and the comprehension of neuropathy: 1) the host immune response, that builds the architecture of the disease forms differentiation and the types of reactions undergoing during the treatment; 2) The disease extension on the nerves and over the skin, expressed as numbers of nerves and skin areas involved and, 3) the temporal aspect, related to the evolution of this highly chronic disease.

The HD neuropathy is primary demyelinating and frequently leads to an extent axonal loss a long its evolution. The bacilli specificity to Schwann cell (SC) membrane and its basal laminin is well known[1,2] and, it's not selective to one fiber modality, the involvement of fibers is universal[3]. It seems to predominate, at the beginning, in the thin fibers because the unmyelinated SC can carry out thin myelinated fibers and several unmyelinated thin axons jointly. The HD neuropathy is temperature dependent, the bacilli grow better and more in areas with lower temperatures, in the skin and nerves. These areas in nerves are in the limbs distal portions and in the anatomical tunnels. The phenomenon epicenter in the nerve trunks is in the anatomical tunnels at elbows, wrists, knees and ankles. It evolves as a multiple mononeuropahy (MM). Even in the skin it behaves like MM and the pictures of polyneuropathy are actually MM. The higher the cell immune response effectiveness is, the more restrict neuropathy is, in the pole TT and BT are seen mononeuropathies, in the patients classified as BB and BV the MM and, patients VV the MM polyneuropathy like[4].

The HD neuropathy has three great periods of evolution: a) the SC parasitization by *Mycobacterium leprae* (ML) in which the clinical expression is the demyelination, observed by the clinical neurophysiologists and by neuropathologists working conspicuously with HD neuropathy, even before the new researches about ML immunospecificity to SC laminin, as remarked in a brief review about neuropathy management during the IX Congress of Hansenology[4] in 1997; b) the acute and subacute periods, during the reactions type 1 and type 2. In this period the symptoms are intense with sensory and motor loss and pain positive symptoms, lastly there are c) the late nerve impairments, caused manly by the intraneural fibrosis, a kind of interstitial neuropathy[4]. All three periods have long duration, which makes the disease actually too much chronic. The discrepancies about if the neuropathy is primarily demyelinating or axonal degeneration in the literature using the CN approach should be due to this extreme chronicity and the patients series studied may be chosen in distinct periods of evolution[5].

In such development, two complication factors must be mentioned: the entrapment neuropathies and neuropathic pain.

In the anatomical tunnels, which have lower temperature, there are higher bacilli populations than other sites added to predisposition to compression. The edema produced acutely during reactions may cause compression, direct effects of pressure, and also focal ischemia in combination may yet play a role in some entrapment neuropathies[6].

The underline mechanisms of neuropathic pain can be outlined as the sensory axonal loss: deafferentation, causing continuous pain, i.e, paresthesias and burning. The extent demyelination process may lead to axonal reflexes - ephatic responses, clinically presented as paroxysmic pain: chock sensation, stings, pin and pinch in discharges. And, in the skin lesions the destruction of the sensory corpuscles innervation structure may cause disesthesias and allodynia in the skin patches[7].

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The author pointed out the positive and negative sensory symptoms, motor signs and nerve palpation features in HD. The author also suggests the follow up strategy using a clinical score (CS) calculated by summation of the results of Visual analog scale (VAS), Nerve palpation (NP), Graded sensory testing (GST) and Voluntary muscle testing (VMT)[5]. The CS can be disposed in a graphic with the drug doses during the treatment that can help the evolution visualization. In each kind of HD neuropathy: type 1 reaction, type 2 reaction, entrapment neuropathy, neuropathic pain and silent neuropathy, one can observe different graphic evolution, that may assist the therapeutic decision. The motor nerve conduction study is also proposed as part of the neurophysiologic evaluation for follow up especially during the reactions in tunnel sites that shows the underline neuropathology adding information to this more complex condition.

Key words: Hansen´s Disease, neuropathy, reactions, entrapment, neuropathic pain, nerve monitoring.

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