

Leber's hereditary optic neuropathy and Multiple Sclerosis: case report.

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Introduction: Leber's hereditary optic neuropathy (LHON) is a mitochondrial disease characterized by visual loss consequent to optic nerve atrophy. It has been theorized that mitochondrial dysfunction could damage muscular and retinal tissues, and also can cause white matter lesions as seen in multiple sclerosis (MS). This association was described in 1992 by professor Harding, but the exact relationship between LHON and demyelinating lesions is poorly understood. **Case:** A 32-year old man presented an abrupt painless loss of vision of his left eye. 5 months later his right eye was affected as well. Central vision was impaired while peripheral vision was partially preserved. General and neurological examination did not provide other clinical signs. A Lumbar Puncture was made. Analysis of cerebrospinal fluid showed oligoclonal bands and an IgG index of 1.354. A brain magnetic resonance (MRI) showed several demyelinating areas as seen in MS. A genetic study was made with 11778GA mutation. There are different mutations related with LHON but this one is associated with the most disabling disease. Successive cerebral MRI showed new periventricular and cervical lesions some of them with gadolinium enhancement. Following MAGNIMS criteria this patient is diagnosed with Radiologically isolated syndrome (RIS). The association between RIS and LHON is called Harding Syndrome. **Discussion:** Harding syndrome has no specific treatment. The controversial point is to add or not immunomodulatory treatment for high risk RIS. Although there is not enough evidence that supports treating these patients, several studies have suggested that a young-age onset, the presence of gadolinium enhancing or medullary lesions could be risk factors of short-time conversion from RIS to MS. Further studies have to be undertaken in order to provide the best therapy to treat promptly these patients.