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Advancement in the Diagnosis and Treatment of Optic Neuropathies

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The aim of this presentation is to discuss the diagnosis, treatment and follow- up of different optic neuropathies. Pictures of new technology devices such as OCT and conventional machines such as Perimetry. The main topic will also be MR images of the orbit and brain, and images of optic nerve problems. There are some new developments in the treatment of optic neuropathies and there will be sharing of different ideas about novel treatments.

Neuropathes: optic neuritis (optic nerve demyelination): sudden, debilitating loss of vision. This occurs primarily in women aged between 18-45 years. Responds well to intravenous steroids at high doses. Approximately 1/3 of the cases of disc edema are seen, and 2/3 are retrobulber. Retrobulber cases move more often toward multiple sclerosis (MS). After acute optic neuritis, the probability of a woman patient developing MS is around 70 per cent in ten years. OCT can detect the development of optic neuritis to MS. Prior ischemical optic neuropathy (non-arteritic): severe, painless vision loss due to a stroke on the optic nerve head. There is Disc edema. In general, patients suffer from hypertension or diabetes. Base in visual environment is altitudinal. No definite treatment and Cup to disc ratio is small. Intravitreal injections (triamcinolone and anti-VEGF) may be tried in acute cases. Traumatic optic neuropathy: Occurs after a direct or indirect trauma to the optic nerve. Steroids are not recommended if there is head concussion.

The optic nerve is the link which transmits visual information from the retina between the eye and the brain. This nerve inflammation is called optic neuritis. Inflammation during optic neuritis can cause damage to the protective sheath (myelin) that covers this nerve and the nerve itself. It can affect one optic nerve or both optic nerves at the same time. Symptoms of optic neuritis can include blurring, blind spots or total vision loss. One may also notice distorted vision, reduced color vision and pain when you move one or both eyes. These types of symptoms may precede loss of vision due to optic neuritis. More commonly the term optic neuropathy describes abnormalities or damage to the optic nerve. This damage could be from blocked blood flow, certain medical conditions or toxic exposure. Optic neuritis is a specific cause of this optic neuropathy.

Toxic optic neuropathy: Central, bilateral visual loss. After methanol toxicity with the combination of erythropoietin and steroids there is a new article regarding progress. Optic radiation neuropathy: Bilateral vision impairment months or years following brain tumor radiation therapy. New reports on vision enhancement with intravenous bevacizumab therapy are available. Painless, bilateral visual loss with central scotomas inherited by the maternal mitochondrial DNA mutations: m.11778, m.14484, m.3460. Idebenone treatment in early stage Leber's disease has been shown to be beneficial. Gene therapy is another option. Chronic relapsing inflammatory optic neuropathy: Steroid sensitive optic neuropathy which recurs after steroid withdrawal. Long term steroids or other immunosuppressive agents are used. There are many other optic neuropathies with infectious, inflammatory etc. etiology which can be discussed, too.

The optic nerve is susceptible to damage from toxins, including drugs, metals (e.g. lead, mercury and thallium), organic solvents (ethylene glycol, toluene, styrene and perchloroethylene), methanol, carbon dioxide and probably some sort of tobacco. This group of disorders is named toxic optic neuropathy (TON) and is characterized by bilateral visual loss, papillomacular bundle damage, central or cecocentral scotoma, and reduction of colour vision. Toxic optic neuropathy might be triggered or just enhanced by nutritional deficits, including the vitamins thiamine (B1), riboflavin (B2), niacin (B3), pyridoxine (B6), cobalamin (B12), folic acid and proteins with sulphur containing amino acids (Phillips 2005). Among malnourished and intoxicated patients, for example, both factors usually play a synergistic role contributing to the TON. Disorders, nutritional optic neuropathy (NON) and TON, are also very similar, and they usually cannot be differentiated based on clinical signs and symptoms. Both disorders are rare in economically developed countries. They are more prevalent in developing countries, because of people's greater exposure to toxic substances in environment and food, and coexisting malnutrition. No racial, gender and age-dependent predilections have been shown.

TON is dose and duration dependent and occurs more often with anti-tuberculosis drugs (ethambutol and isoniazid), some antimicrobial agents (linezolid, ciprofloxacin, cimetidine and chloramphenicol), antiepileptic drugs (vigabatrin), disulfiram (for chronic alcoholism), halogenated hydroquinolones (amebicidal medications), antimetabolites (e.g. methotrexate, cisplatin, carboplatin, vincristine and cyclosporin), tamoxifen and sildenafil. Severe poisoning causes nausea, vomiting and abdominal pain and affects the central nervous system in a similar way as ethanol.

Toxic and nutritional optic neuropathies are often classified as acquired mitochondrial optic neuropathies overlapping with congenital mitochondrial optic neuropathies, including LHON and dominant optic atrophy (DOA).

Leber hereditary optic neuropathy (LHON) is an inherited sort of vision loss. Although this condition usually begins during a person's teens or twenties, rare cases may appear in infancy or later in adulthood. For unknown reasons, males are affected far more often than females.

Blurring and clouding of vision are usually the primary symptoms of LHON. Such vision issues may occur in one eye or in both eyes simultaneously; if the loss of vision begins in one eye, the opposite eye is usually affected within several weeks or months. Over time, vision in both eyes worsens with a severe loss of sharpness (visual acuity) and chromatic vision. This condition mainly affects sight, which is required for detailed tasks like reading, driving, and recognizing faces. Vision loss results from the death of cells within the nerve that relays visual information from the eyes to the brain (the optic nerve). Although sight gradually improves during a small percentage of cases, in most cases the vision loss is profound and permanent.

Vision loss is usually the sole symptom of LHON; however, some families with additional signs and symptoms are reported. In these individuals, the condition is described as "LHON plus." additionally to vision loss, the features of LHON plus can include movement disorders, tremors, and abnormalities of the electrical signals that control the heartbeat (cardiac conduction defects). Some affected individuals develop features almost like MS, which may be a chronic disorder characterized by muscle weakness, poor coordination, numbness, and a spread of other health problems.