MILLER FISHER SYNDROME WITH OPTIC NEURITIS

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Abstract:
Optic neuritis can occur in a patient suffering from Miller Fisher Syndrome resulting in acute visual loss. Only very few such cases have been reported so far in literature. A 40 years old lady presented with the classic triad of Miller Fisher Syndrome (ataxia, external ophthalmoplegia and areflexia). She developed sudden total visual loss in right eye followed by left eye with bilateral optic disc edema which was proved to be papillitis with fluorescein fundus angiography. MRI Brain with contrast was normal and CSF was acellular with elevated protein and normal glucose level. She was treated with steroids in her vision. After 4 weeks of treatment Ataxia and ophthalmoplegia improved but there was no significant improvement in her vision.

Keyword:
Miller Fisher Syndrome, optic Neuritis, ataxia, external ophthalmoplegia, areflexia Miller fisher syndrome with optic neuritis

Case History:
40 years old lady from Vellore presented to our hospital in July 2010 with history of holocranial headache which was continuous not associated with vomiting, photophobia or phonophobia, swaying side to side while standing and walking and clumsiness of both upper limbs, bilateral ptosis and inability to roll both eyes of 2 weeks duration. Then she developed sudden onset visual loss in right eye followed by vision loss in left eye after 1 week. She was admitted in our hospital. There was no history of fever, altered sensorium, redness of eyes, eye pain, other cranial nerve symptoms, motor weakness, sensory symptoms, bowel or bladder involvement.
Physical examination:
Higher mental function was normal. She had no perception of light in right eye and was able to perceive hand movements in left eye. She had bilateral optic disc edema with peripapillary haemorrhages. She had bilateral ptosis and total external ophthalmoplegia. Other cranial nerves were normal. Motor examination showed normal tone, muscle power and areflexia with flexor plantar. Sensory examination was normal except for mild decrease in vibration below both knees, and negative Romberg’s sign. Cerebellar examination showed dysmetria, intention tremor in both upper limbs, gait ataxia and inability to do tandem walking.

Investigations:
Blood counts, blood sugar, renal and liver function tests were normal, MRI Brain and spinal cord with contrast with MR Angiogram and Venogram was normal. CSF was acellular with elevated protein (78mg/dl) and normal glucose level. Nerve conduction Study showed normal motor conduction and absent Sural sensory potentials. Visual Evoked Potentials showed prolonged P100 latency in left eye and absent waveform in right eye. fluorescin Fundus angiogram showed early dye leakage in the optic disc and peripapillary region.

Neuroophthalmologist’s opinion was obtained and the diagnosis of optic neuritis was confirmed. The patient was treated with intravenous methyl prednisolone 1gm intravenous once daily for 7 days followed by oral prednisolone for 1 month in tapering doses with two weeks of treatment the patient’s eye movements started improving and become near normal. After 1½ months ataxia and upper limb clumsiness improved and her gait become normal by 1 month. static during follow up. She did not have recur-

Her visual acuity did not improve and remained static during follow up. She did not have recurrence of the illness.

Discussion:
Miller fisher syndrome is considered to be a variant of Guillain Barre’s Syndrome accounting for 5% of cases and is characterized by ophthalmoplegia, ataxia and areflexia. Ocular signs range from complete ophthalmoplegia including unreactive pupils, or external ophthalmoplegia with or without ptosis. Cases can overlap with Guillain Barre’s Syndrome and present with quadriaparesis. Electrodiagnostic studies demonstrate axonal process involving sensory fibres with mild motor conduction abnormalities. Brain stem MRI scans do not demonstrate brainstem or cerebellar lesions. Serum IgG antibodies to the ganglioside GQ1b are found in acute phase sera of most parients with MFS and GBS with ophthalmoplegia, which suggests that the antibodies are disease-specific and related to the pathogenesis. Our case has the classic triad of Miller fisher syndrome with normal MRI of brain and spinal cord with CSF showing albumino cytological dissociation and nerve conduction studies showing absent sural sensory potentials. Our case developed sudden onset bilateral visual loss, with bilateral disc edema and with VEP and fluorescin angiography features of optic neuritis. Very few such case reports of Miller fisher syndrome with optic neuritis has been reported in literature. Such a rare presentation should be thought of in appropriate clinical setting for prompt treatment.
References: