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FOSTER KENNEDY SYNDROME OR NOT ?

Ophthalmology

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ABSTRACT

Foster Kennedy Syndrome is characterised by insidious reduction in vision in one eye, accompanied by clinically significant papilledema in the fellow eye in presence of mass lesion on ipsilateral side. There are three types – 1.Optic atrophy in ipsilateral eye and papilledema in fellow eye (most common type). 2 .Bilateral papilledema with unilateral optic atrophy. 3. Bilateral papilledema developing into bilateral optic atrophy.

KEYWORDS

Foster Kennedy Syndrome, Papilloedema, Optic Atrophy

Case Report: -

27 years old female complaining of gradual, progressive diminution of vision in left eye and headache since 2 years. She had history of double vision for 1 month associated with pain on eye movements 2 years back. MRI revealed subtle signal abnormality in the left optic nerve suggestive of left optic neuritis. Small soft tissue is noted along the left cavernous sinus reaching the left orbital apex likely suggestive of Tolosa Hunt Syndrome. And she was started on IV MPS and then tapered on Tab. Prednisolone. MRI was repeated after 6 months which was suggestive of reduced in volume involving intraorbital segments of left optic nerve with thinning of perineural CSF sleeve likely left optic nerve atrophy.

Table 1: Ocular examination

Right eye		Left eye
6/9(-1.0 DS)	Vision	Within normal limits
Within normal limits	Colour vision	Within normal limits
Normal size, Reactive to light	Pupils	Direct- mid dilated, non reactive to light. Swinging flash light- Relative afferent pathway defect.
Within normal limits	Rest anterior segment	Within normal limits
Media is clear, 360° blurring of disc margins obliteration of cup disc ratio, hyperemic disc, engorgement of blood vessels, macula normal, Foveal reflex present	Fundus	Media clear, pale disc, attenuated blood vessels, macula normal, Foveal reflex present
Enlargement of blind spot	Visual fields	Total absolute scotoma

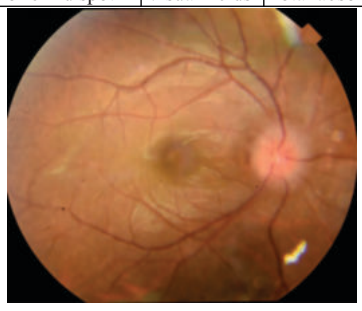

Image 1: Fundus photo of right eye

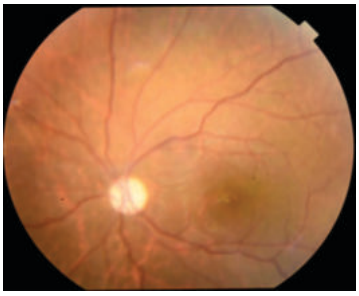

Image 2: Fundus photo of left eye

Recent MRI revealed nodular pachymeningeal thickening along the posterior aspect of falx and tentorium cerebelli likely suggestive of inflammatory etiology. Right optic nerve shows mild hyperintense signals. Loss of flow void is seen in posterior aspect of superior sagittal sinus, left transverse sinus likely suggestive of thrombosis due to pachymeningeal thickness. Considering history and neuroimaging, this is a case of Pseudo Foster Kennedy syndrome and NOT Foster Kennedy Syndrome. Cause for raised ICT in this patient could be pachymeningitis leading to decreased CSF absorption for which neurologist has advised pachymeningeal biopsy which was suggestive of vasculitis and she was started on oral steroids.

DISCUSSION:

Foster Kennedy syndrome[1] leads to visual loss due to compression of optic nerve causing its atrophy in one eye and papilledema in the contralateral eye due to increased intracranial pressure. The underlying cause of this condition is the presence of an intracranial space-occupying lesion that compresses the ipsilateral optic nerve, compressive optic atrophy. The lesion can also cause increased intracranial pressure, thus leading to papilledema in the fellow eye found. Meningiomas are the most common type of tumors found in the brain of patients with Foster Kennedy syndrome. There are typically three types of Foster Kennedy syndrome, based on whether or not the atrophy and papilledema are unilateral or bilateral. Type 1 is the most frequent presenting form with optic atrophy in the ipsilateral eye and papilledema in the contralateral eye. Type 2 is characterized by bilateral papilledema and unilateral optic atrophy. Type 3 is defined by bilateral papilledema developing into bilateral optic atrophy. The three types of Foster Kennedy syndrome are caused by different stages of metastasis of brain tumors. The management of Foster Kennedy syndrome is usually by surgical resection, although chemotherapy and radiotherapy are also employed, especially in old geriatric patients who are more prone to mortality.

Pseudo-Foster-Kennedy Syndrome[2] is characterized by unilateral optic atrophy with contralateral optic disc edema in absence of intracranial mass causing compression of optic nerve. The most common etiology for Pseudo-Foster-Kennedy Syndrome is bilateral sequential anterior ischemic optic neuropathies, either arteritic or non-arteritic anterior ischemic optic neuropathy. Non-arteritic anterior ischemic optic neuropathy is more common. Patients can present with new onset, acute visual loss and optic disc swelling in one eye with prior visual loss and secondary optic disc atrophy resulting from a previous ischemic event in the other eye .Other causes of Pseudo-Foster-Kennedy Syndrome are diseases that cause optic atrophy from a prior event with an acute episode of optic disc edema like prior papillitis and secondary optic atrophy from infectious, inflammatory, infiltrative,or other cause.Treatment of Pseudo-Foster-Kennedy Syndrome depends on etiology like for arteritic anterior ischemic optic neuropathy,high dose systemic corticosteroid therapy is given and for optic neuritis, intravenous methylprednisone followed by oral prednisone could be considered.

Tolosa hunt syndrome[3] is idiopathic inflammation in the region of the cavernous sinus and/or superior orbital fissure, causing painful cranial neuropathies. Patient presents with severe and unilateral periorbital headache associated with painful and restricted eye movements. Hence, tolosa hunt syndrome is also known as painful ophthalmoplegia, recurrent ophthalmoplegia, ophthalmoplegia syndrome.

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