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Optic neuritis followed by tuberculosis in a case of systemic lupus erythematosus

KEYWORDS: Optic neuritis, Systemic lupus erythematosus, Tuberculosis

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ABSTRACT *A 17 year old female patient presented to us with sudden blurring of vision in left eye since 3 days. She was diagnosed previously with systemic lupus erythematosus and tuberculosis.*

Optic neuritis in systemic lupus erythematosus is very uncommon(1%).1 There are very few case reports in India. Unilateral optic neuritis associated with systemic lupus erythematosus in a child which occurs after treating tuberculosis makes this case important to report.

Systemic lupus erythematosus is a systemic disease with multiorgan involvement. Ocular features in systemic lupus erythematosus are rare compared to other systems. Early diagnosis and intervention will give good visual prognosis.

Case presentation:
A 15 year old female presented with sudden blurring of vision in left eye since 3 days along with history of fever and joint pains. On examination her left eye best corrected visual acuity was 20/80(Snellen chart) with gradeII relative afferent papillary defect. Colour vision testing revealed red green deficiency. Amsler grid test revealed no abnormality. Contrast sensitivity testing was decreased. Fundus examination showed optic disc swelling of 2DD and haemorrhages over the disc.VEP revealed a delayed P100 response with increased latency in left eye. Visual field examination revealed an enlargement of blind spot . Right eye examination was completely normal.

Systemic examination revealed joint pains and butterfly rash. Three years before she was diagnosed as a case of systemic lupus erythematosus and treated with steroids. Later she developed pulmonary tuberculosis for which ATT was used for 9 months. ATT medication was stopped 1 year ago following which X ray chest postero anterior view revealed no abnormality and acid fast bacilli samples were negative. Cardiac examination showed minimal pericardial effusion. Magnetic resonance imaging was normal. Anti dsDNA and ANA are positive.

Patient was started on intravenous methyl prednisolone 1gm for 3 days followed by 11 days of oral prednisolone. Fundus examination after 2 days revealed reduction of disc swelling and haemorrhages.

At 15 days followup her visual acuity was 20/20 with resolved optic disc edema.

Figure1at the time of presentation showing optic disc edema

Figure2 after 15 days of presentation showing resolved disc edema

Discussion:
Systemic lupus erythematosus (SLE) is a chronic, autoimmune, multisystem disease which may affect the eyes and/or visual system in up to a third of patients. Although early recognition and treatment have led to a reduction in severe ocular complications, ocular involvement in systemic lupus erythematosus is still a potentially blinding condition.[1]

The diagnosis of Systemic lupus erythematosus is essentially clinical and is generally based on the presence of four or more of the 11 features listed by the American College of Rheumatology classification criteria (ACR/ ARA).[2] Ophthalmic problems may be an important part of overall disease activity, and are thus featured in the the British Isles Lupus Assessment Group index of disease activity (BILAG2004).[3] Ocular manifestations in systemic lupus erythematosus are fairly common, potentially sight threatening and may be the presenting feature of their disease. Systemic lupus erythematosus may affect almost any part of the eye and visual pathway.

Optic nerve disease occurs in around 1% of patients with systemic lupus erythematosus, and includes acute retrobulbar optic neuritis, papillitis, anterior ischemic optic neuropathy, posterior ischemic optic neuropathy or slow progressive visual loss (singerman & jampol 1991). Presenting visual acuity in systemic lupus erythematosus associated optic neuropathy is poor with most patients seeing worse than 20/200.[4] The increased severity of disease in systemic lupus erythematosus-associated optic neuritis compared to idiopathic optic neuritis stems from differences in pathogenesis. Systemic

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lupus erythematosus-optic neuritis is not due to a primary inflammatory demyelinating process but rather an ischemic process that can cause subsequent demyelination and axonal necrosis. The degree of axonal loss correlates to visual outcome.electron microscopic studies showed increased expression of glial fibrillary acidic protein in glial end feet which leads to vasculitis, haemorrhaging and axonal degeneration suggests impaired circulation to optic nerve and retina[6]. In the absence of other features of systemic lupus erythematosus, it can be very difficult to distinguish this from the typical optic neuritis of demyelinating disease. Acute optic neuritis may also be bilateral and associated with transverse myelopathy.[6] In our case patient presented to us with painless loss of vision and not associated with transverse myelopathy.

Visual recovery in SLE optic neuritis may range from full recovery to count fingers vision. In a study by lin et al only 50% of patients recovered to better than 20/25, while 37.5% maintained a visual acuity worse than 20/200.[4] There have been some reports regarding the beneficial effect of intravenously administered "pulse" methylprednisolone in autoimmune optic neuropathy.[8] In our case also intravenous methyl prednisolone followed by oral prednisolone gave good result. In some studies it is proved that intravenous cyclophosphamide showed good results.[9]

Another significant finding in our case is, she suffered from tuberculosis after she was diagnosed as a case of systemic lupus erythematosus. The high incidence of serious infections is one of the most disturbing problems in the management of patients with systemic lupus erythematosus. Besides immunosuppressive therapy several immunological abnormalities appear to contribute to the pathogenesis . Hence such patients are at increased risk of viral, mycotic and other opportunistic infections. Our case used anti tuberculosis therapy for 9months. No study has reported onset after withdrawal of ethambutol.[10]These anti tuberculous drugs such as ethambutol and isoniazid may cause optic neuritis. It is nonetheless rare in patients prescribed standard doses. Retrobulbar neuritis is the most common, with involvement of either axial fibres or, less commonly, periaxial fibres.

Presenting ocular symptoms vary among affected individuals. Patients may complain of bilateral progressive painless blurring of vision or decreased colour perception. Central vision is most commonly affected, though other visual field loss has also been described. Some individuals may be asymptomatic with abnormalities detected only by vision tests. The findings of physical examination are likewise variable. Both eyes are usually symmetrically affected, if any abnormality is detected. The pupils may be bilaterally sluggish to light with no relative afferent pupillary defect. Visual acuity drop varies greatly from nil or minimal reduction to no light perception. Central scotoma is the most common visual field defect, but bitemporal defects or peripheral field constriction have been reported. Dyschromatopsia (abnormal colour perception) may be the earliest sign of toxicity, classically documented to be red-green colour changes. In contrast to this, the report by Polak et al stated that blue-yellow defects were the most common and early defect in patients without any visual symptoms. Nonetheless the subtle blue-yellow defects could only be detected using the generally unavailable desaturated panel of Lanthony and not Ishihara charts nor Farnsworth-Munsell D-15 test. Fundoscopic examination is usually normal. In our case we excluded this as she discontinued tuberculous medication 1 year back. One of the principal theories for its toxicity has been the zinc-chelating effect of ethambutol and its metabolite. Postulated biochemical pathways that mediate the toxic damage include downstream effector caspase-3 and caspase-6, and an excitotoxic pathway.[10]

According to conclusion of results in optic neuritis treatment trial, It was evident that intravenous methylprednisolone followed by oral prednisone speeds the recovery of visual loss due to optic neuritis and results in slightly better vision at 6 months, compared with the placebo group.[11] However, the results of this study could not be applied directly to the cases of autoimmune neuropathy because the patients who had clinical evidence of a systemic disease other than multiple sclerosis were excluded. Further study is needed to identify the optimal therapy for optic neuritis in systemic lupus erythemotosus.

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