PAPILLEDEMA IN SYSTEMIC LUPUS ERYTHEMATOSUS: A CASE REPORT

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BACKGROUND

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease with multisystemic involvement, including a variety of ocular manifestations. In fact, eye involvement related to SLE can be diagnosed in approximately one third of patients. Alterations can include the posterior segment triggering retinopathy, vasculitis and even neuro-ophthalmological alterations. This latter condition includes optic disc edema secondary to high intracranial pressure (ICP) and papilledema is one of the neuro-ophthalmic clinical findings in SLE. There are several causes of ICP which can lead to papilledema, including idiopathic intracranial hypertension, space-occupying lesions, cerebral edema, among others.

CASE REPORT

32-year-old, woman diagnosed with SLE 14 years ago. Her clinical lupus manifestations were articular and cutaneous involvements with photosensitivity and lupus nephritis (ISN/RPS Class IV). Her relevant labs were positive ANA Hep-2 homogeneous pattern 1:320, anti-Smith, anti-double stranded DNA (anti-dsDNA) and negative antiphospholipid. She was initially treated with cyclophosphamide (CP). Thereafter, she used for maintenance mycophenolate mofetil with sustained remission. She has a past medical history of controlled systemic arterial hypertension and bilateral hip osteonecrosis. Recently, she had a flare with high protein-creatinine ratio, low complement, active urine sediment and positive anti-dsDNA. The patient accepted to participate in a lupus nephritis trials, which comprised an extensive ophthalmological evaluation, independent of clinical symptoms, prior to induction treatment with methylprednisolone and CP indicated for lupus nephritis. She had no visual complaints. On examination, the best visual acuity of right eye was 0.8 and in the left eye was 1.0. Intraocular pressure was 18 mmHg in both eyes. The anterior chamber was normal and the fundus examination showed a nasal swelling of the optic disk bilaterally. A spectral-domain optical coherence tomography was performed and confirmed the presence of edema (Figs. 1 and 2). Central nervous system resonance and liquor analysis were unremarkable. A panel of infectious causes were ruled out and the finding was interpreted as related to active disease. We speculated that an antibody mediate process directed to arachnoid villi may underlie our findings. The patient underwent lupus nephritis induction treatment and the optic disk swelling showed total improvement right at the first treatment month.

Figure 1. Optical coherence tomography – right (A) and left (B) optic nerves with blurred disc margins suggestive of optic disc edema.

Figure 2. Optical coherence tomography – cross section of the right (A) and left (B) optic nerves at the levels of the green arrows in Fig. 1, showing an increase in the thickness of the nerve fiber layer (blue arrows) suggesting edema in nasal topography.

CONCLUSION

Asymptomatic optic disc edema in SLE is a rare condition with great potential for irreversible eye damage. The additional observation of an exclusive association with disease activity and not with thrombosis, emphasizes the relevance of regular ophthalmology assessment in this disease, particularly during flares.

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