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Case report

Central serous chorioretinopathy as a cause of vision loss in chronic relapsing inflammatory optic neuropathy

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ABSTRACT

Purpose: Chronic relapsing inflammatory optic neuropathy (CRION) is a type of idiopathic recurrent optic neuritis that responds to systemic corticosteroids and relapses on steroid withdrawal or dose reduction. Central serous chorioretinopathy (CSCR) is often associated with glucocorticoid therapy. This paper aims to highlight CSCR as a cause of visual loss in patients being treated with corticosteroids for optic neuritis.

Observations: We describe the case of a 42-year-old woman with a history of CRION in her left eye who presented with painful vision loss in the right eye and diffuse right optic disc edema. Steroid therapy was initiated, leading to visual and perimetric improvement. Two months later however, the patient returned with painless visual loss, now related to CSCR. Despite oral steroids being continued, there was spontaneous tomographic and visual recovery after four months.

Conclusions and importance: We believe this is the first report of CSCR causing vision loss in a patient with CRION treated with oral corticosteroids. CSCR should be suspected in patients with optic neuritis of any cause who develop vision loss while on treatment with steroids.

1. Introduction

Chronic relapsing inflammatory optic neuropathy (CRION) is a form of idiopathic recurrent optic neuritis that responds promptly to treatment with systemic corticosteroids and relapses upon withdrawal or reduction of the dose of steroids.[1–3] Central serous chorioretinopathy (CSCR) is an underdiagnosed condition characterized by macular neurosensory retinal detachment and/or detachment of retinal pigment epithelium.[4] Traditionally, it has been associated with hypercortisolism, most commonly arising from glucocorticoid therapy.[5,6] We describe a previously unreported case of a CRION patient who presented with CSCR while she was being treated with steroids.

2. Case report

A 39-year-old woman was first admitted to our hospital with recurrent episodes of subacute deterioration of vision in her left eye (OS), accompanied by pain on eye movement, left relative afferent pupillary defect and left optic disc edema. Her visual acuity was 20/16 in the right eye (OD) and 20/70 OS. Standard automated perimetry showed a left inferior altitudinal defect.

A complete workup for optic neuritis was undertaken. Her complete blood count, hepatic, renal, and thyroid function, erythrocyte sedimentation rate, C-reactive protein, serum angiotensin-converting enzyme, antinuclear antibodies, anti-neutrophil cytoplasmic antibodies, toxoplasmosis screen, and cytomegalovirus, Epstein-Barr virus, herpes simplex virus, varicella zoster virus, Brucella, *Borrelia burgdorferi*, *Treponema pallidum*, QuantiFERON®-TB Gold assay, human immunodeficiency virus, and hepatitis B and C virus serology results were normal. NMO-IgG testing was negative and there were no oligoclonal bands on analysis of cerebrospinal fluid. Magnetic resonance imaging of the brain and orbits showed enlargement of the left optic nerve, consistent with left optic neuritis, but was otherwise normal. There were no pulmonary or interstitial lesions on chest computed tomography (CT) or gallium scans.

The patient was treated with intravenous and then oral methylprednisolone, showing prompt pain resolution and visual improvement. However, her vision deteriorated and her ocular pain recurred whenever the oral steroids were tapered, and a diagnosis of CRION was made.

The patient was lost to follow-up at that time but re-presented to the emergency department three years later, at the age of 42 years, with a

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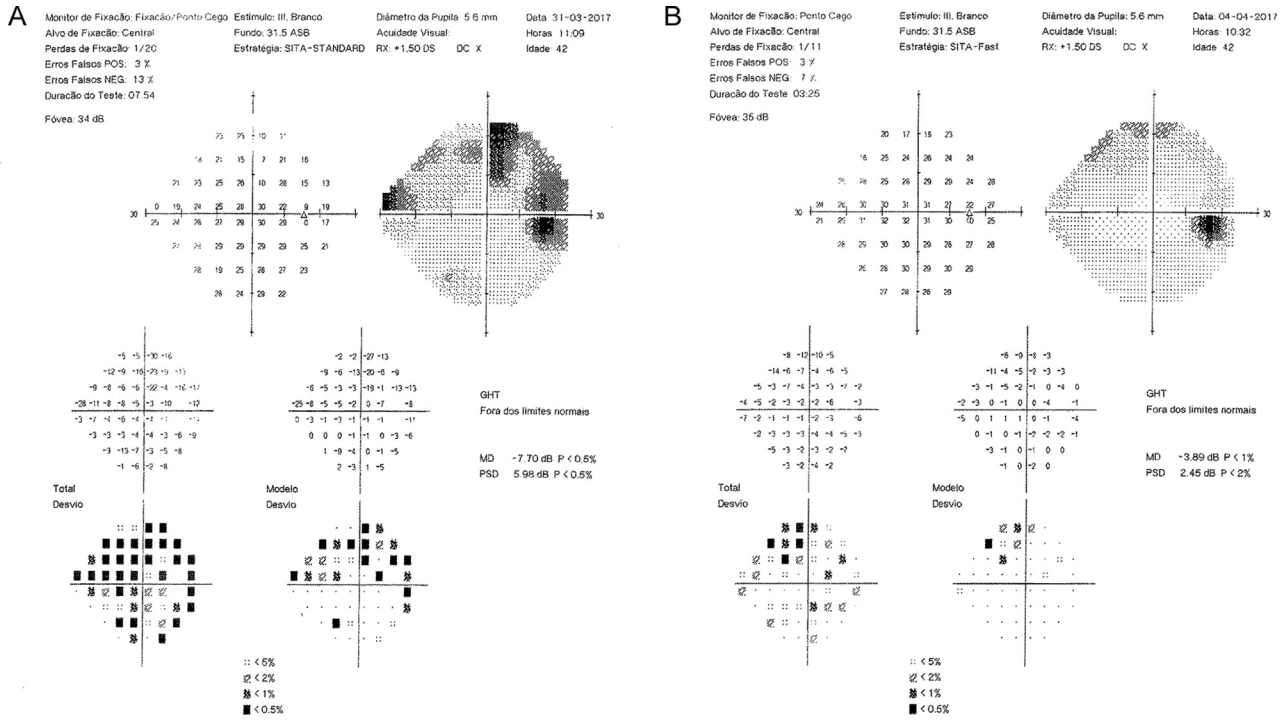

Fig. 1. Standard automated perimetry (24-2) during a relapse of CRION in the right eye, showing a superior arcuate defect(A), and after 3 days of intravenous methylprednisolone, showing marked perimetric improvement (B).

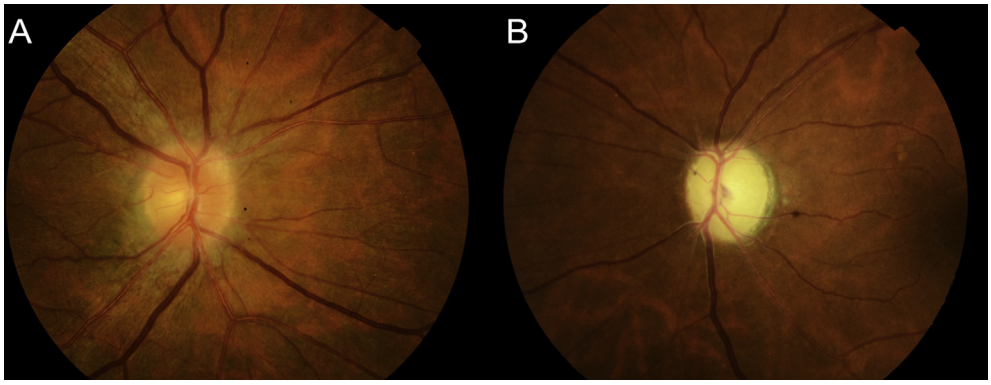

Fig. 2. Fundus photography during a relapse of CRION in the right eye, showing diffuse optic disc edema OD (A) and optic disc pallor OS (B).

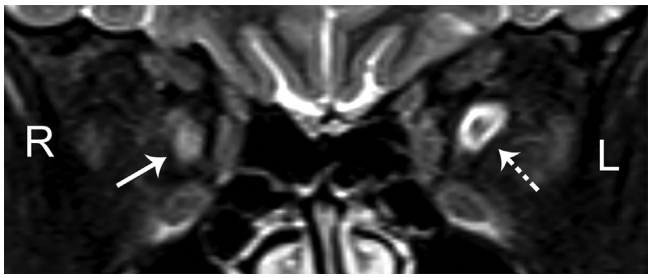

Fig. 3. T2-weighted magnetic resonance image of the orbits (coronal view). There is marked homogeneous hyperintensity of the right optic nerve (solid arrow) and an increased volume of cerebrospinal fluid surrounding the left optic nerve, consistent with optic neuritis OD and optic nerve atrophy OS (dashed arrow). R, right; L, left.

3-day history of painful vision loss in the OD. On examination, her visual acuity was 20/20 OD and her color vision was 17/17 Ishihara plates. Standard automated perimetry showed a superior arcuate defect (Fig. 1A). She had no light perception OS and there was a relative afferent pupillary defect OS. Fundoscopy showed diffuse optic disc edema OD and optic disc pallor OS (Fig. 2). Neurologic examination and systems review were normal. Magnetic resonance imaging of the brain and orbits revealed thickening and enhancement of the right optic nerve and significant atrophy of the left optic nerve, but was otherwise normal (Fig. 3). The patient was admitted and treated with intravenous methylprednisolone. Within 3 days, she noticed prompt resolution of the pain and there was improvement on sequential standard automated perimetry (Fig. 1B). Optic disc edema improved after treatment with intravenous steroids and she was discharged and maintained on oral prednisone 60 mg/day on a slow tapering regimen, oral methotrexate 15 mg/week, folic acid 1 mg/day, and omeprazole 40 mg/day.

Two months later, the patient reported painless worsening of vision in her right eye. Visual acuity was 20/25 OD and there was no dyschromatopsia or optic disc edema OD. A shallow neurosensory macular detachment was suspected on fundoscopy (Fig. 4A) and confirmed by spectral-domain optical coherence tomography (SD-OCT, Fig. 5A). There were no other fundus abnormalities. Thus, a diagnosis of CSCR