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Vitamin deficiency optic neuropathy - a case report

Neuropatia óptica carencial - relato de caso

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KEYWORDS:

Optic Nerve Diseases; Vitamin B-Complex; Toxic Optic Neuropathy.

ABSTRACT

Nutricional Optic neuropathy is a rare cause of visual deficits, associated with restrictive diets, pernicious anemia, alcoholism, and others. It has a good prognosis if diagnosed and treated properly. The present study presents a case of a 27-year-old female patient with a diagnosis of vitamin deficiency optic neuropathy, which was treated with B-complex vitamin replacement.

PALAVRAS CHAVES:

Doenças do Nervo Óptico; Complexo Vitamínico B; Neuropatia Óptica Tóxica.

RESUMO

Neuropatia óptica carencial é uma causa rara de déficit visual, associada a dietas restritivas, anemia perniciosa, alcoolismo, entre outras. Possui bom prognóstico quando diagnosticada e tratada corretamente. O presente trabalho apresenta caso de paciente do sexo feminino, 27 anos, apresentando sintomas compatíveis e posterior diagnóstico de neuropatia óptica carencial, sendo tratada com reposição de vitaminas do complexo B.

INTRODUCTION

Optic nerve atrophy associated with nutritional deficiency is an uncommon cause of painless, progressive, subacute, bilateral, symmetrical visual loss which can be difficult to diagnose. There are loss of contrast sensitivity, severe and early dyschromatopsia, and central or centrocecal scotoma[1]. The disease is caused by a lack of copper or B-complex vitamins, especially cyanocobalamin (B12), thiamine (B1), and folic acid (B9)[2]. Its incidence has been increasing due to the growing number of bariatric surgeries and strict vegetarian diets[3]. In addition, patients with pernicious anemia and alcoholics can also be deficient in B12. Vitamin B12 is present in animal food sources as well as nutritional formulas. The pathogenesis is unknown, but it is believed to be related to a failure in mitochondrial oxidative phosphorylation, with reduced ATP (adenosine triphosphate) production and free radical accumulation[1]. Optic disc changes include mild edema, moderate hyperemia, hemorrhage, temporal disc pallor, thinning of the nerve fiber layer of the papillomacular bundle, and optic atrophy[4]. The pupillary light reflex may be slightly reduced[5]. This pattern resembles Leber's hereditary neuropathy and toxic as well[6]. The treatment is based on the replacement of B-complex vitamins. Early recognition and diagnosis are vital to start the treatment and avoid irreversible optic nerve atrophy[2].

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CASE REPORT

A 27-year-old female presented with a complaint of progressive bilateral low visual acuity, associated with difficulty focusing and central scotoma in the left eye (OS). The patient had hypothyroidism and used Levothyroxine. She was a non-smoker and reported rare alcoholic intake. She had been on a vegetarian diet for the last 8 years, adopting the vegan diet 3 years ago, on a regimen of manipulated vitamin B12 500 IUI/day.

On ophthalmological examination, she presented corrected visual acuity of 20/30 in the right eye (OD) and 20/50 in the left eye (OS). Pupils were photoreactive, with mild relative afferent pupillary defect in OS. Mildly color vision defect (Hardy Rand Rittler test) in both eyes were noted. Biomicroscopy had no changes. Intraocular pressure was 12 mmHg in both eyes. Fundoscopy showed sharp-edged optic disc, mild temporal pallor in the OD, moderate temporal pallor in OS, reduced retinal nerve fiber layer in papillomacular bundle, and normal macula were also observed (Figure 1). Optical coherence tomography showed decreased temporal nerve fiber layer and reduced macular ganglion cell layer (Figure 2). 30-2 visual fields showed cecocentral scotoma (Figure 3).

Figure 1. Color retinography shows temporal optic disc pallor in both eyes.

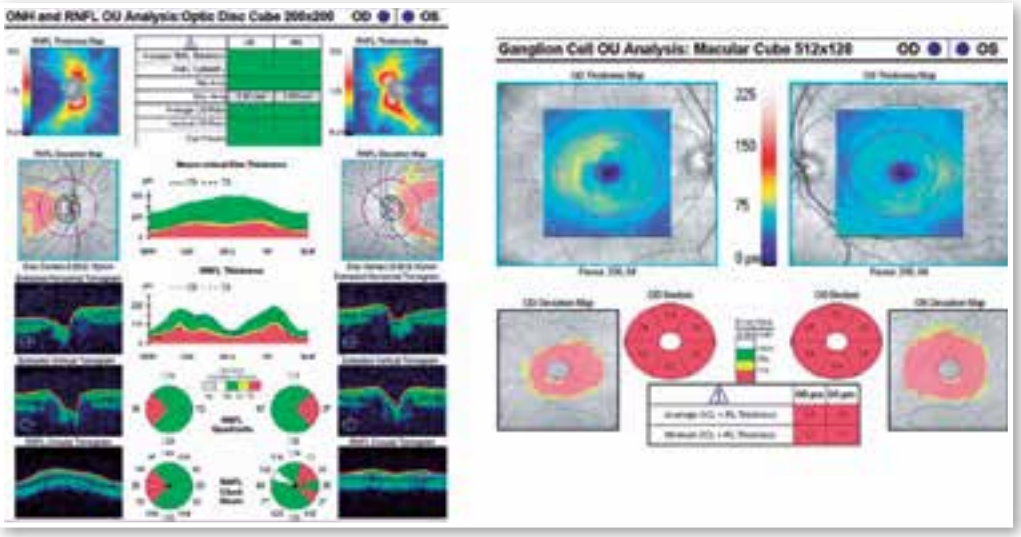

Figure 2. Optical coherence tomography shows reduction of the temporal nerve fiber layer in both eyes and reduction of ganglion cells thickness in macular topography.

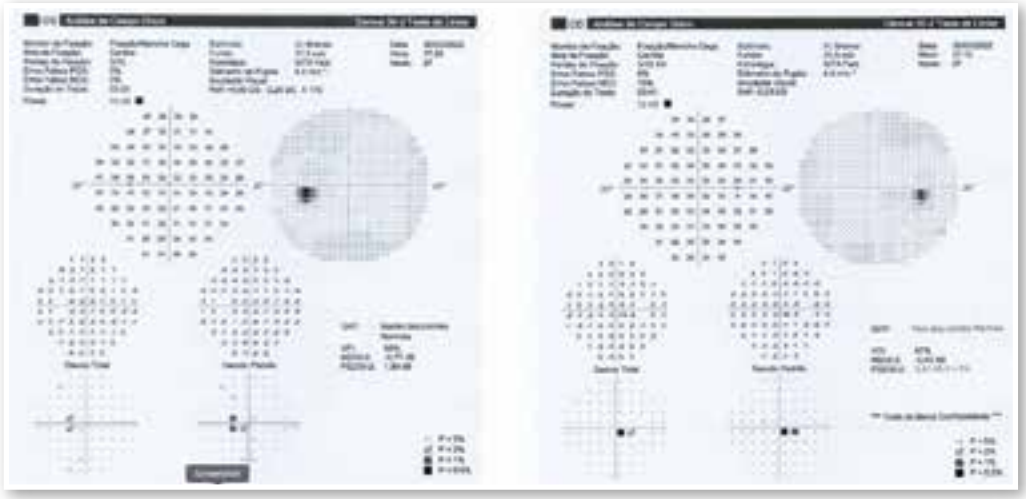

Figure 3. Visual field shows bilateral cecocentral scotoma.

The hypothesis of nutritional optic neuropathy was formulated, discussed with the patient, and it was decided to start vitamin B1 100 mg + B6 100 mg + B12 5,000 IUI, 3 times/week, intramuscular.

DISCUSSION

Optic neuropathy deficiency is a rare but possibly misdiagnosed cause of bilateral, relatively symmetrical, painless, progressive visual loss caused by B1 complex nutrient deficiency. Since it has a good prognosis if diagnosed and treated properly, its early recognition is crucial[6]. Currently, due to the increase in the number of bariatric surgeries and restricted diets, cases of vitamin deficiency optic neuropathy have increased[7,8]. This neuropathy can also be caused by pernicious anemia or alcohol consumption[1]. It initially presents as dyschromatopsia, loss of contrast sensitivity, and central or cecocentral scotomas, bilaterally and relatively symmetrically[4]. Physical examination shows an edematous and hyperemic optic disc in the early stages, evolving with possible temporal pallor due to loss of nerve fibers from the papillomacular bundle[6]. Additional tests include optical coherence tomography showing reduction in the retinal nerve fiber layer and ganglion cell complex, prior to the appearance of fundoscopic changes[1] and visual fields with central or cecocentral defects[9]. The treatment consists on B vitamin supplementation, which has proven to be effective[2]. Serum vitamin B12 and folate levels do not necessarily reflect the tissue amount of these nutrients, and therefore may be normal even in the presence of reduced tissue levels (functional deficiency of B12 or folate). Therefore, early empirical treatment is suggested in cases of strongly suspected vitamin deficiency optic neuropathy, even in patients with normal serum levels of vitamins, in order to avoid permanent neurological damage[6].

The presented patient had a typical presentation of vitamin deficiency optic neuropathy, due to a strict vegetarian diet. The findings of complementary exams confirmed the hypothesis, and she was submitted to treatment with B vitamin supplementation.

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