Decreased visual acuity secondary to celiac disease

Baixa acuidade visual secundária à doença celíaca

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Cranial computed tomography showing posterior cortical-subcortical calcifications in the occipital and occipitoparietal regions

The male patient was a 24-year-old student who was a native of São Paulo (SP). The onset of left hemibody numbness associated with decreased strength and difficulty speaking was 15 days ago. It progressed with decreased bilateral visual acuity. After 90 minutes, the patient presented with photophobia and severe holocranial headaches, which were unresponsive to common analgesics. The headaches improved after analgesic medication, but the visual change persisted. For his past medical history, the patient has had recurrent episodes of "sparkles" in his vision that progressed to headaches since he was 12 years of age. He was evaluated by a neurologist at that time and showed changes in the central nervous system, but no specific diagnosis was indicated. The patient reported the occurrence of four additional episodes over the past year that were similar to the current episode, although the intensity of the headaches was lower. In his family history, there have been cases of migraine headaches. Since the last episode, the patient has been taking valproic acid and dexamethasone. An ophthalmologic examination showed a best-corrected

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visual acuity of 0.1 in both eyes. The remainder of the examination consisted of external ocular motility, biomicroscopy, Goldmann applanation tonometry and neuromotor reflexes, which were all unchanged in both eyes. A cranial computerized tomography scan was performed, which showed posterior cortical-subcortical calcifications in the occipital and occipitoparietal regions. The patient was then referred for an evaluation to the gastroenterology service in the hospital, where he underwent a gastrointestinal endoscopy including a small bowel biopsy and received a definitive diagnosis of celiac disease. After eight months on a gluten-free diet, the patient showed improved visual acuity (1.0 in both eyes).

In 2004, Zelnik et al.⁽¹⁾ identified the co-existence of neurological symptoms in 51.4% of patients in a pediatric population with celiac disease compared to 19.9% in the control group. In a case report of a 4-yearold patient presenting with epileptic seizures, Lea et al.⁽²⁾ documented the progressive appearance of bilateral parietal intracranial calcifications and a subsequent diagnosis of celiac disease, which resulted in adequate seizure control without the need for medication after instituting dietary changes. The description of the triad of bilateral parietal calcifications, epilepsy and celiac disease is already widely reported in the literature⁽³⁻¹⁰⁾. Pfaender et al.⁽⁴⁾ reported the cases of three patients whose initial symptoms of disease were the occurrence of various visual symptoms, which were sometimes followed by significant headaches/migraines and the identification of bilateral parietal intracranial calcifications, with a subsequent diagnosis of celiac disease. This description reinforces the initial proposal that the occurrence of paroxysmal visual manifestations is the primary characteristic of epilepsy arising from the occipital lobe, despite being a rare focal form. The three patients showed an improvement of symptoms after instituting a restrictive diet and antiepileptic medication.

The mechanisms involved in the onset of intracranial calcifications specifically localized to the bilateral parietal region have not yet been elucidated. *Post-mortem* analysis of brain tissue from some patients drives the suspicions that immune-mediated phenomena may alter the cerebral vascular permeability, favoring the localized deposition of calcium^(3,6).

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