Intravitreal Triamcinolone Acetonide for Macular Edema in HLA-B27 Negative Ankylosing Spondylitis

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Abstract
We report a case of a human leukocyte antigen B27 (HLA-B27)-negative patient with cystoid macular edema (CME) and ankylosing spondylitis (AS) after treatment with triamcinolone acetonide. The patient complained of deterioration of visual acuity of the right eye during the last 10 days. At presentation visual acuity of the right eye was 0.2, and the ophthalmic examination did not reveal any sign of active uveitis. Fluorescein angiography (FA) and ocular coherent tomography (OCT) showed CME. The left eye was normal with a visual acuity of 0.9. Eight weeks after intravitreal injection of triamcinolone acetonide, visual acuity improved to 0.8 and OCT revealed regression of macular edema. Six months later no recurrence was observed. Our case report indicates for the first time that CME may occur in AS independently of the presence of HLA-B27 and intraocular inflammation. Intravitreal use of triamcinolone acetonide can reduce macular edema and restore visual acuity.

Introduction

Ankylosing spondylitis (AS) is an important condition to diagnose in patients with uveitis and it has a long-established association with the human leukocyte antigen B27 (HLA-B27). 80–95% of the patients with AS are HLA-B27 positive [1, 2]. It must be stated that, among patients with uveitis in whom HLA-B27 is detected, the rate of AS is 41% compared with 0% in the HLA-B27 negative patients [3]. We present our case because to the best of our knowledge this is the first patient with uveitis and HLA-B27 negative AS. Furthermore, the good therapeutic result after intravitreal use of triamcinolone acetonide is signalled.
Case Report

A white 52-year-old female was referred with visual acuity deterioration in the right eye (RE) for the previous 4 months. She had a medical history of AS and was in treatment with oral indomethacin 150 mg/day and sulfasalazine 2 g/day. Diagnosis of AS had been made 10 years ago, almost 6 years after the first rheumatologic symptoms emerged. The patient was tested negative for HLA-B27. Her ophthalmologic history included only 1 episode of anterior uveitis in the RE almost 3 years ago for which she had received oral corticosteroid prednisone for a period of 6–8 weeks. Since then the patient had had no other episode of anterior uveitis and treatment with corticosteroids had not been repeated until now.

Best-corrected visual acuity (BCVA) of the RE was 0.2 and of the left eye (LE) 0.9. Slit-lamp examination revealed only a minor subcapsular posterior cataract RE > LE and no evidence of signs of anterior uveitis bilaterally (cells: 0 and Tyndall: –). The fundus examination disclosed cystoid macular edema (CME) without cells in the vitreous in the RE. The LE was normal. Fluorescein angiography and optical coherence tomography confirmed the diagnosis of CME (fig. 1a, c, e). After discussion with the patient, the decision for intravitreal injection of triamcinolone acetonide in the RE was made.

Eight weeks after the injection, CME resolved significantly (fig. 1b, d, f) and BCVA improved to 0.8. At the end of the follow-up 6 months later, no signs of recurrence were observed and BCVA remained 0.8 in the RE.

Discussion

AS is a chronic rheumatic disease with a reported prevalence ranging from 0.2 to 1.1%. The disease is characterized by chronic inflammation, bone destruction and aberrant bone repair, thereby resulting in severe movement impairment and disability [4]. Human leukocyte antigen-typing has documented a significant association between the genetic marker HLA-B27 and AS [5].

The most common ocular manifestation of AS is anterior uveitis which may lead to severe complications such as diffuse vitritis, papillitis, retinal vasculitis and CME [6]. Linssen and Meenken [7] documented that the prevalence of anterior uveitis in patients with HLA-B27 positive AS is significantly higher compared to those with HLA-B27 negative AS.

Almost 13.4% of the patients with HLA-B27-associated anterior uveitis develop CME. Uy et al. [8] reported that the prevalence of CME is 5 times more frequent in anterior uveitis with HLA-B27 haplotype compared to idiopathic HLA-B27 negative uveitis.

However, there are no available data in the literature regarding the incidence of ocular complications in HLA-B27 negative anterior uveitis, particularly in patients with AS and HLA-B27 negative uveitis, since the co-existence of these diseases is extremely rare.

To the best of our knowledge, this is the first patient with AS and HLA-B27 negativity who developed CME in the absence of chronic anterior uveitis. CME was successfully treated with intravitreal injection of triamcinolone acetonide and visual restoration was achieved with no signs of recurrence in a 6-month follow-up. It seems that even patients with HLA-B27 negative AS may manifest severe ocular complications, such as CME, independently from the presence of chronic anterior uveitis. Therefore, a regular ophthalmological examination of these patients is important. Intravitreal injection of triamcinolone acetonide instead of Avastin or other anti-VEGF could be a therapy approach worth considering in such cases, as it might facilitate CME regression and improvement of visual acuity.
Disclosure Statement

The authors report no conflict of interest.
Fig. 1. Optical coherence tomography (OCT) before intravitreal injection of triamcinolone acetonide showing macular edema with typical cystoid hyporeflective spaces in the outer retinal layers (a). OCT 8 weeks after intravitreal injection of triamcinolone acetonide showing significant resolution of macular edema (b). Multiple areas of macular hyperfluorescence and fluorescein leakage in the early (c) and late (e) phase of fluorescein angiography showing evidence of CME at presentation. Significant reduction of hyperfluorescence and leakage indicating improvement of the angiographic findings in the early (d) and late (f) phase, 8 weeks after intravitreal injection of triamcinolone acetonide.
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