ABSTRACT

Case report: We report the case of a 76-year-old woman who attended our hospital because of a sudden loss of visual acuity in her left eye. The problem appeared to be that of retrobulbar optic neuritis. However, the age of the patient together with the simultaneous use of Infliximab to treat her rheumatoid arthritis, suggests the demyelination may have been associated with the Infliximab use.

Discussion: TNFα inhibitors (including Infliximab) are associated with demyelinating diseases including optic neuritis (Arch Soc Esp Oftalmol 2007; 82: 109-112).

Key words: Optic neuritis, Infliximab, TNFα inhibitors, multiple sclerosis, demyelination.

RESUMEN

Caso clínico: Se describe el caso de una paciente de 76 años de edad que presenta una disminución brusca de la agudeza visual en su ojo izquierdo. El cuadro es compatible con una neuritis óptica retrobulbar que en el contexto de una paciente de esta edad y en tratamiento con Infliximab por una artritis reumatoide sugiere la posibilidad de que se trate de una desmielinización asociada al tratamiento con Infliximab.

Discusión: Los inhibidores del TNFα (entre ellos el Infliximab) pueden dar lugar a eventos desmielinizantes entre los cuales puede encontrarse una neuritis óptica retrobulbar.

Palabras clave: Neuritis óptica, Infliximab, inhibidores del TNFα, esclerosis múltiple, desmielinización.
INTRODUCTION

Retrobulbar optical neuritis are a retrobulbar inflammation of the optic nerve, with demyelinating diseases being the most frequent etiology, and within these, multiple sclerosis. Other diseases that can give rise to demyelinating conditions are infections, vasculitis etc. Recently, it has been demonstrated that the a (TNFα) inhibitors of the tumor like necrosis factor also could be the cause of optical neuritis (1).

CASE REPORT

A 76 year-old woman who attended the urgency ward of our hospital exhibiting abrupt visual acuity reduction in her left eye with 24 hours of evolution. In her personal history she emphasizes the presence of a long-standing rheumatoid arthritis which had required treatment with Metotre xate and which at the present time was being treated for 20 months with Infliximab (Remicade®). She did not exhibit ophthalmological antecedents of interest.

The initial ophthalmological exploration was as follows: corrected visual acuity in right eye (RE): 1,0 and left eye (KE): 0,15; relative afferent pupillar defect in left eye; biomicroscopy of the anterior segment showed bilateral corticonuclear cataracts; and in the eye fundus hard drusen were appraised on vascular arches as well as a macula and optic nerve with normal appearance. Accordingly, analyses were ordered including globular sedimentation speed (VSG) at the urgency ward. These gave normal results. Subsequently, a Humphrey 30-2 campimetry was performed, revealing a generalized depression of sensitivity in the left eye, with the right field of vision being normal (fig. 1). The Farnsworth-Munsell colors test of the left eye exhibited an alteration at the level of the yellow blue axis.

As we suspected an injury involving the left prechiasmatic visual pathway, a skull-orbitary nuclear magnetic resonance was requested which revealed multiple demyelinating injuries of the subcortical and periventricular white substance with involvement of the callous-septal union (figs. 2 and 3). Considering the irregular morphology of the injuries and their characteristic location, and in spite of the age criterion, the first possibility we considered was multiple sclerosis.

Fig. 1: Generalized reduction of sensitivity in left eye.

Nevertheless, and after consulting with neurology, we considered the possibility of retrobulbar optical neuritis with demyelinating etiology in relation to the treatment with Infliximab. It was decided to suspend the treatment with Infliximab and establish treatment with 1g pulses of methylprednisolone for 3 days. The patient responded with a clear recovery of the visual acuity (1,0) as well as the disappearance of the campimetric scotoma in the successive controls and to date (fig. 4).

DISCUSSION

The TNFα tumor like necrosis factor is a cytokine derived from the macrophages that stimulate inflammatory processes by means of joining the cellular surface receptors. Infliximab is an IgG type antibody that ligates to the TNFα inhibiting its activity.
Since the introduction of anti TNFα for the treatment of rheumatoid arthritis and Crohn’s disease, its use has extended as immunomodulating treatment for different self immune diseases, becoming a standard medication for these patients.

In spite of the high security that this drug exhibits in diagnosed rheumatoid arthritis patients, adverse respiratory and neurological effects have been described, including two cases of exacerbation of preexisting demyelinization in patients treated with Infliximab and who previously exhibited a rapidly progressing multiple sclerosis (2). In 2002 Foroozan et al (1) described the first case of retrobulbar optic neuritis of demyelinizing etiology in a 55 year-old patient with rheumatoid arthritis who was in treatment with Infliximab.

Regardless of the possibility that it was possible that this might be an accidental finding, the authors emphasize the need to be alert about the possibility of finding demyelinizing events in patients who are

Fig. 3: Demyelinizing injuries in the NMR cross section.

Fig. 4: Recovery of the left field of vision after suspension of treatment with Infliximab.
being treated with TNFα inhibitors. This recommendation is based both on the similarity of this case with cases of exacerbation of multiple sclerosis after treatment with infliximab (2) and on the results obtained in tests with Lenercept (another TNFα inhibitor) in patients with multiple sclerosis, where a greater tendency has been observed towards the exacerbation of the disease in these patients in comparison with placebo groups (3). Subsequently, new cases of demyelinizing neuritis have been described in patients with other pathologies (Crohn’s disease) who had received treatment with Infliximab (4) as well as some cases of bilateral anterior optic neuropathy related to treatment with Infliximab.

Due to the etiopathogenic factors which relate TNFα inhibitors to the appearance of demyelinating events as well as the advanced age of our patient, in whom a first occurrence of multiple sclerosis is very infrequent, we considered that this is a case of demyelination associated to Infliximab treatment.

In order to conclude, we would like to point out the possibility of finding in the future conditions similar to that which we have shown and the need to suspend medication with TNFα inhibitors (5) in these cases, due to the increasing popularity of these drugs in our hospitals.

REFERENCES