of the Spectralis OCT device was significantly negatively correlated with disease duration (−0.551; p < 0.001). Other tests revealed only a slight correlation.

In conclusion, we found significant correlations between visual function tests and RNFL thickness with the severity of AD (measured by disease duration). The correlations between the MMSE score and different visual function tests, however, were slight. Colour vision impairments are strongly correlated with disease duration; at this point, therefore, colour vision impairment could be a good biomarker for diagnosis and follow-up of AD. Gundogan et al. (2007) also found that colour vision assessment is better correlated than RNFL thickness with disease activity in other neurodegenerative diseases, such as multiple sclerosis. We only recruited patients with mild to moderate AD, so more studies including patients with mild cognitive impairment and severe AD and with a larger sample size are necessary. Visual function tests, especially colour vision assessment, seem to be more related with neurodegenerative disease progression than other digital imaging techniques such as OCT. Visual impairments are often among the earliest complaints of patients with AD; accordingly, visual function tests (especially colour vision assessment) could facilitate evaluation of suspected AD.

References


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In reply to the comment of Dr Asaf Achiron to a paper: Circulating anti-retinal antibodies in response to anti-angiogenic therapy in exudative age-related macular degeneration

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Editor,

I have read with interest the important paper by Kubicka-Trzaska et al. (2014). My concern is about the statistics presented. Throughout the manuscript, the significant values of correlations between antiretinal antibodies and various clinical parameters were denoted, but the strengths of these correlations were not. Reporting the correlation strength is essential in accurately interpreting data.

Reference


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Editor,

We appreciate Dr Asaf Achiron (2015) comments on statistics presented in the article Kubicka-Trzaska et al. (2014). Indeed, we found no significant correlations between antiretinal antibodies (ARAs) types and choroidal neovascularization (CNV) diameter, central retinal thickness (CRT) and best corrected visual acuity (BCVA) in AMD patients treated with intravitreal injections of bevacizumab. No statistically significant associations between the number of ARAs immunofluorescence patterns, and all of these clinical parameters of disease activity were observed either. However, we found a tendency indicating that the multitude immunofluorescence patterns of ARAs were observed in sera of patients with CNV diameter ≥ 3000 μm and CRT ≥ 300 μm. It is worth to emphasize that the complexity of circulating ARAs immunofluorescence patterns above three did not influence significantly disease activity.

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References


Late posterior segment relapses in a series of Vogt–Koyanagi–Harada disease

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Editor,

We recently reported that 73.8% of subjects diagnosed with Vogt–Koyanagi–Harada (VKH) disease experienced recurrences (Errera et al. 2011). Based on our experience, we believed that finding an exudative retinal detachment (ERD) that responded to corticosteroids or other medical treatment more than 1 year after onset is low. To better evaluate late posterior recurrences, we reviewed the records of a large series of 45 patients with VKH disease from four tertiary centres. The median follow-up duration was 34 months since initial presentation.

Late posterior segment relapses were defined as posterior segment manifestations of inflammation occurring later than 52 weeks after first presentation of VKH (Garcia-Valdes et al. 2000; Sachdev et al. 2008; Dolz-Marco et al. 2011). The study adhered to the tenets of the Declaration of Helsinki.

Relapsing ERDs were noted within 10 months after presentation in 16 (84%) of 19 subjects who experienced such reactivation, with only four experiencing late ERDs, one of whom also had early relapses. Report of four subjects with late ERDs relapses at a median of 19 months (16–25 months).

By examination and optical coherence tomography (OCT), subretinal fluid was limited localized areas of submacular ERDs in all late posterior segment relapses (Fig. 1). Intravenous pulse therapy was used within the first month for three of the four subjects who experienced a late posterior reactivation. In the fourth subject, the diagnosis of VKH disease was made 3 months after the first inflammatory signs, which caused a delay in the initiation of the correct treatment. This latter subject had treatment with corticosteroids for only 1 month, while the other three subjects received an initial duration of systemic corticosteroids for at least 6 months.

In all four subjects, immunosuppressive therapy (infliximab, cyclosporin or interferon) was initiated after the first relapse because response to therapy with corticosteroid at acceptable long-term doses was insufficient.

All four subjects had been on immunomodulatory agents (cyclophosphamide, infliximab and azathioprine), but these agents had been stopped 5 to 13 months prior to the relapse.

In one subject, the relapse occurred while he was taking 25 mg/day of prednisone. He had responded to reintroduction of high-dose corticotherapy for an episode 6 months previously, and he was on tapering of therapy. The second subject was receiving both prednisone and cyclophosphamide. The third was on infliximab and low-dose oral corticosteroids at the time of relapse.

In the experience at our institutions there were 4 late relapses (ERDs) of 45 subjects with an incidence rate of 12 cases per 1000 VKH affected person-years. Late relapses as ERDs occurred in patients receiving early intravenous pulse corticosteroids, and all four had been on other corticosteroid-sparing immunomodulatory therapy at some point. One possible explanation is that the need for more aggressive treatment in our patients with late relapses of VKH may be a marker of more severe disease. Early corticosteroid-sparing immunomodulatory therapy might be useful as first-line therapy to reduce the development of complications (Abu El-Asrar et al. 2013). Although it is difficult to conclude because we do not have enough statistical power (tests of Kaplan-Meier), we observed that subjects who had a longer duration of high dose corticosteroids (>3 months) at presentation of the disease were more likely to present posterior segment reactivation than those with shorter duration of corticosteroid therapy (This comparison raises the issue of resistance to corticosteroid treatment (and/or resistance to immunosuppressive therapies), of various therapeutics.

We found, however, that late ERDs are limited to local submacular ERDs in our patients; larger ERDs do seem to be very rare. And if found, alternative diagnoses such as idiopathic choriotoretinopathy (CSR) or rheumatogenous detachment need to be considered. Conversely to what has been reported in CSR, no pigment epithelial detachment was seen on OCT in our cases. In all four subjects, ERD resolved following an increase or reinitiation of corticosteroids therapy which is another argument for excluding CSR.

Annexes

‘Chronic’ or ‘recurrences’ are terms referring to subjects being off treatment