Letters to the Editors

Ocular involvement in giant cell arteritis

Sirs,

Giant cell arteritis (GCA) represents the most common primary vasculitis of the elderly that usually involves large- and medium-sized arteries (1). The wide spectrum of clinical manifestations can extensively vary from cranial symptoms, such as headache, jaw claudication or visual alterations, to constitutional symptoms, like fever, weight loss or asthaenia (2, 3). Temporal artery biopsy (TAB) and starting glucocorticoids (GC) therapy still represent the gold standard recommendations to early diagnose and treat the disease (3-5). Permanent, partial or complete, visual loss in one or both eyes is one of the most dreadful manifestations of GCA and has been observed in about 20% of patients in most series (6,7). Ocular symptoms can vary from amaurosis fugax to transient diplopia and to sudden unilateral or bilateral visual loss. In the majority of cases, impaired vision is caused by arteritic anterior ischaemic optic neuropathy, which is related to an occlusive involvement of the posterior ciliary arteries (1); moreover, less commonly, visual loss can also be due to central retinal artery occlusion, retrobulbar optic neuritis, or posterior ischaemic optic neuritis. In the present study we analysed the frequency of ocular involvement in a group of GCA patients. Out of a cohort of more than 237 GCA, 127 biopsy proven patients (25 males and 102 females, mean ± SD age: 79±2 years; mean ± SD age at the onset 72±6 years, mean disease duration 7 years) were studied. The primary aim of the study was to evaluate the prevalence of impaired vision as first clinical symptom at the onset of disease in a cohort of patients with GCA. The secondary aim was to compare the subset of patients characterised by the presence of ocular involvement with those who did not experience impaired vision. Prevalence of ocular involvement demonstrated by ophthalmological examination and/or visual field and/or fluorescein-angiography and/or optical coherence tomography were retrospectively analysed in all patients. The study was approved by the local ethics committee of the University of Pisa (Italy). Forty-two percent (n=72) of patients presented at the onset of disease visual alterations. The most common presenting symptoms among patients who experienced ocular involvement were: visual loss of varying severity (94%), amaurosis fugax (24%), diplopia (10%) and eye pain (4%). Anterior ischaemic optic neuropathy was observed in 71% of the patients, while central retinal artery occlusion was revealed in 17% of cases. In all patients with permanent visual loss (n=12), fundoscopic examination showed signs of ischaemic optic neuropathy; moreover, among these, only 4 patients were affected by permanent bilateral visual loss. No epidemiological difference was noted between the groups of patients who experience impaired vision and those who did not, except for the mean age at disease onset that was higher in patients with ocular impairment (75±5 *vs*. 70±7 years). From a serological point of view, GCA patients with ocular involvement were characterised by ESR and PCR median values lower than in patients without visual impairment ($p<0.001$; $p=0.002$). Regarding the timing of treatment, ninety percent of patients with ocular involvement received high doses of steroids (Intravenous 6-methyl prednisolone 125–250 mg for 3 days, followed by step-by-step reduction to oral steroid therapy) within two weeks from the onset, while 6% of subjects received high doses of GC after 2 weeks and within 1 month from the onset. Moreover, in 4% of the cases GC was started after 1 month from the onset of ocular symptoms. Notably, all patients treated with GC after 2 weeks from the onset were characterised by permanent visual loss. According to the literature data, results from our cohor confirm that ocular involvement is frequent in GCA biopsy proven patients. Moreover, as reported from other studies (5-6), GCA patients with ocular involvement seem to be characterised by ESR and PCR values lower than in patients without visual impairment. Unfortunately, once visual loss is established, it is usually permanent and this risk makes GCA an ophthalmological emergency. Many studies have unequivocally demonstrated that GC therapy is able to prevent visual loss, making an early recognition and treatment features of primary importance.

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