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LETTER TO THE EDITOR

Reply to: "Bilateral Non-arteritic Anterior Ischaemic Optic Neuropathy as the Presentation of Systemic Amyloidosis"

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We read with interest the article by Kanaan et al. entitled "Bilateral non-arteritic Anterior Ischaemic Optic Neuropathy as the presentation of systemic amyloidosis". In their excellent and well-documented paper, the authors report a case of a 75-year-old woman affected by bilateral sequential optic neuropathy, in whom a diagnosis of giant cell arteritis was suspected based on fluorescein angiography and temporal artery duplex ultrasound features. However, temporal artery biopsy was consistent with amyloidosis. The authors concluded that "In this case, bilateral sequential ischaemic optic neuropathy mimicking non-arteritic anterior ischaemic optic neuropathy (AION) was the presenting sign of systemic amyloidosis involving the temporal arteries". They also report that the patient had a history of "fully investigated" bilateral idiopathic intermediate uveitis, diagnosed 30 years previously and that did not require treatment for the preceding 2 years. Despite this, the patient had required systemic immunosuppression to control her intermediate uveitis. There is no report of the clinical features of the intermediate uveitis, such as the appearance of the vitreous.

Amyloidosis is a heterogeneous group of disorders characterized by the deposition of hyaline extracellular material. Virtually any structure of the eye and adnexa may be involved.[1]

Vitreous amyloidosis is a rare condition that mainly occurs in Familial Amyloidotic Polyneuropathy,[2] although isolated disease has been reported.[3,4] Vitreous opacities in amyloid disease may be the only sign of amyloidosis, and present years before other eye or systemic features develop. They must not be confused with those resulting from more common causes such as haemorrhage, inflammation, degeneration, or neoplasms.[5] Opacities associated with amyloid disease are usually bilateral but may be asymmetric or unilateral. They are classically described as "initially granular with wispy fringes", growing in size and aggregating to form a "glass wool appearance".

Inflammation from intermediate uveitis may present a confusing picture, particularly in the case of old quiescent vitritis with vitreous debris.[6]

Definitive diagnosis of any ocular lesion requires confirmational biopsy and specific histological examination (e.g. positive Congo red staining). Vitreous samples obtained during vitrectomy may thus be useful in establishing the diagnosis of amyloidosis.

Thus in our opinion, before concluding that the bilateral AION was the presenting sign of systemic amyloidosis, the intermediate uveitis should have been investigated through pars plana vitrectomy. Potentially, in reported case, the vitritis could have been the real presenting ophthalmic feature of amyloidosis. The clinical value of the paper, however, remains unchanged: amyloidosis must be considered in the differential diagnosis of giant cell arteritis since it has the potential to cause jaw claudication and AION.

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Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the article.

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