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CORRESPONDENCE

Sir,

Ranibizumab for subfoveal choroidal neovascularization in Bietti crystalline retinopathy

Bietti crystalline retinopathy was first described by Bietti[1] and is characterized by the presence of numerous yellow glistening intraretinal crystals in the posterior pole and tapetoretinal degeneration. Crystalline corneal deposits are associated in about one-third of patients but are not required for clinical diagnosis. Bietti disease is a rare genetic disorder with autosomal recessive inheritance involving *CYP4V2* gene mutation.[2] The natural course of the disease leads to progressive visual loss due to the atrophy of the retinal pigment epithelium and the choroid. Choroidal neovascularization (CNV) is unusual. Here, we report a case of macular CNV in Bietti retinopathy treated with intravitreal injections of ranibizumab.

Case report

A 29-year-old woman with unremarkable medical and family history reported a progressive vision loss in her left eye (LE) for 5 days. Visual acuity was 20/20 in the right eye and 20/50 in the LE. Fundus examination revealed numerous yellow glistening retinal crystals scattered throughout the posterior pole and the mid-periphery in both eyes. No corneal abnormalities were detected. Diagnosis of Bietti crystalline retinopathy was established based on fundus examination and fluorescein and ICG-SLO angiographies and OCT.[3] Visual field, electroretinogram, and electrooculogram were normal. CNV in the LE was seen on fluorescein and ICG angiographies. OCT confirmed the intraretinal exudation (Figure 1). Intravitreal injections were performed after informed consent in agreement with French legislation and our local ethics committee. After three injections of ranibizumab, visual acuity was 20/32 in the LE and no recurrence was noted after 6 months of follow-up (Figure 2).

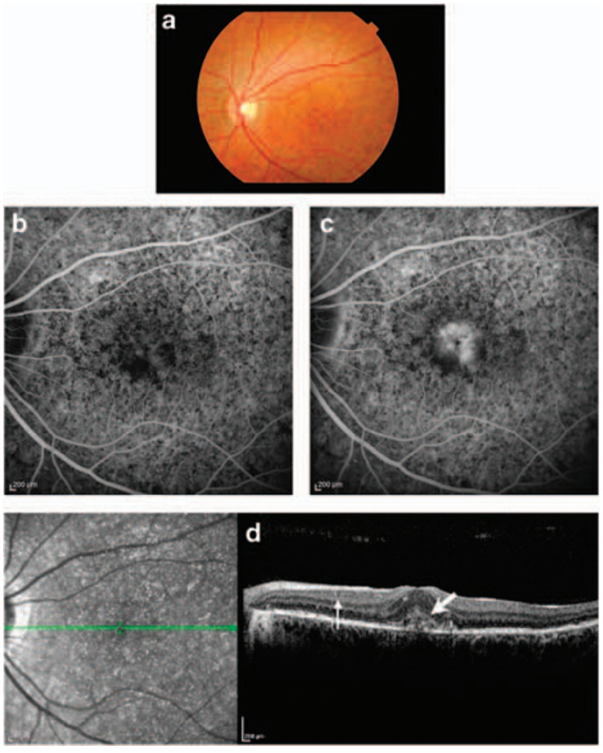

Figure 1 Initial examination. (a) Fundus photograph shows numerous glistening crystalline deposits throughout the posterior pole. Fluorescein angiography at first examination revealed a choroidal neovascular membrane with central hyperfluorescence in early transit phase (b) and leakage in the late phase (c). (d) Spectral domain OCT showed a subfoveal hyperreflective pre-epithelial lesion and macular oedema. Thin white arrow shows the localization of crystalline deposits. Large white arrow shows the localization of choroidal new vessels.

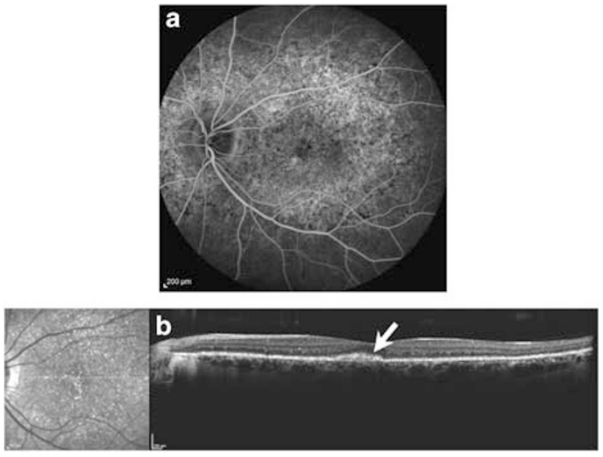

Figure 2 After three intravitreal injections of ranibizumab in the LE, (a) fluorescein angiography shows tiny late inferior hyperfluorescence due to the impregnation of fibrosis scar without leakage. (b) OCT reveals hyperreflective pre-epithelial spindle corresponding to the fibrosis of the CNV without intra- or subretinal leakage (large white arrow).

Comment

One case of peripapillary CNV[4] has been previously described in Bietti retinopathy, but to our knowledge, it is the first description of subfoveolar CNV. The mechanism of occurrence of CNV in Bietti retinopathy remains unclear. CNV can occur at early stages. In this peculiar case, intravitreal injections of ranibizumab allowed good visual recovery of the acuity without any adverse side effect. However, antiangiogenic agents should be administered carefully in young patients and after complete medical information.

In summary, subfoveolar CNV can occur in Bietti retinopathy. Intravitreal ranibizumab can be considered as a therapeutic option.

Conflict of interest

The authors declare no conflict of interest.

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Sir,

Trichotillomania following herpetic neuralgia

A 58-year-old gentleman presented to the eye casualty complaining of reduction in vision in his right eye. He had been diagnosed with right trigeminal nerve (ophthalmic branch) herpetic zoster by his general practitioner 1 month ago, and at that time he had completed an oral course of acyclovir. History was otherwise unremarkable.

Acuity was 6/18 in the right eye and 6/5 in the left. His right-sided trigeminal shingles rash was now only just visible. His right cornea had reduced sensation and there was a rapid tear break-up time on this side. The corneal epithelium was intact but irregularly heaped. Ocular examination was otherwise unremarkable.

A diagnosis of post-herpetic, neurotrophic corneal epitheliopathy was made. Regular lubricants were applied and a lower punctual plug was inserted. Within 6 weeks the corneal epithelium was healthy and vision returned to 6/5.

The gentleman had also reported post-herpetic neuralgia, which in this case had an interesting manifestation. He had found relief (and almost gratification) from this trigeminal neuralgia by recurrently pulling his right eyebrow. As a result, he had substantial unilateral eyebrow loss (see Figure 1).

There are over 30 reported causes for eyebrow loss,[1] one of which is trichotillomania (TTM). TTM is defined as the compulsion to pull out one's own hair. TTM is an impulse control disorder wherein hair is pulled out by the patient, typically from the scalp but also from the eyebrows and eyelashes.[2] TTM is often found to co-exist with mood and anxiety disorders and is classified by the *Diagnostic and Statistical Manual of Mental Disorders*.[3]

This gentleman had no associated psychiatric disorder, but he had developed a marked and persistent impulse to repeatedly pull his right eyebrow, resulting in a cosmetically undesirable consequence of unilateral eyebrow loss. This significantly affected his quality of life and he became keen for treatment. Treatment for TTM includes various drugs, and also forms of cognitive behavioural therapy, such as habit-reversal training.[2] This patient's neuralgia is now controlled with gabapentin.

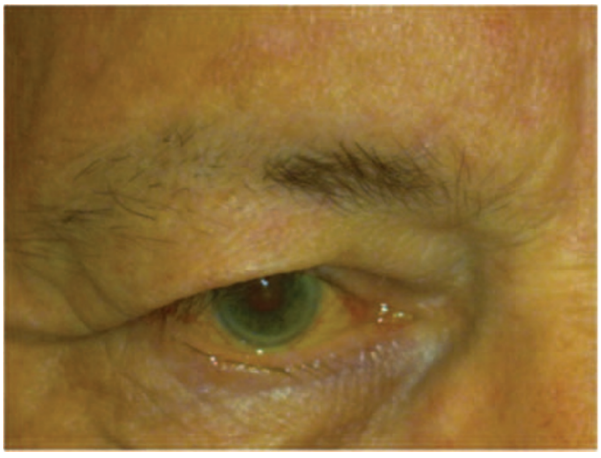

Figure 1 Loss of the right eyebrow (trichotillomania following herpetic neuralgia).

To the author's knowledge, this is the first case reporting eyebrow loss due to TTM following herpetic neuralgia.

Conflict of interest

The author declares no conflict of interest.

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