attributable to intraocular inflammation, such as in VKH syndrome.[1,2] Although symptomatic inflammatory conditions may prompt examination and discovery of choroidal hypopigmentation, primary choroidal vitiligo may go undiagnosed for years because these patients are usually asymptomatic.

Primary choroidal vitiligo has been described in only a few patients,[3,4,6] all of whom also had cutaneous vitiligo, although skin findings were subtle in some cases.[6] All previous reports of choroidal vitiligo described large patches of choroidal depigmentation.[3–6] Vingerling et al.[3] described 2 Asian patients with cutaneous vitiligo who were found to have primary choroidal vitiligo. No retinal pigment epithelium (RPE) changes were noted. It was proposed that the absence of RPE changes in these patients reflects the different embryological origins of RPE cells (neuroectoderm) and choroidal melanocytes (neural crest).[3]

When large patches of depigmentation occur, primary choroidal vitiligo can mimic choroidal nevi. Shields et al.[6] describe 4 cases of choroidal vitiligo in patients who were referred for evaluation of large choroidal nevi. All 4 patients were darkly pigmented, and the authors believe the normal choroid appeared abnormal compared with the lighter areas of hypopigmentation. These patients demonstrated intact retina and RPE on OCT in the areas of hypopigmentation, and fundus autofluorescence did not demonstrate any evidence of RPE abnormalities. FA showed slight choroidal hyperfluorescence, suggesting a variation of a "window defect" caused by lack of pigmentation in that area.

In our case, the patient presented with multifocal small patches of choroidal depigmentation, which appeared similar to multifocal choroiditis. The patient only revealed his history of cutaneous vitiligo upon questioning after fundoscopic examination. Cases such as this one make the diagnosis of primary choroidal vitiligo particularly difficult, as the patches of depigmentation were not as large as those reported previously and mimicked multifocal choroiditis. Choroidal vitiligo should be included on the differential diagnosis for multifocal depigmented choroidal lesions, particularly in patients with a history of cutaneous vitiligo.

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Erlotinib-associated bilateral anterior uveitis: resolution with posterior sub-Tenon's triamcinolone without erlotinib cessation

Erlotinib (Tarceva; Genetech USA, Inc, San Francisco, Calif.) is an inhibitor of epidermal growth factor receptor tyrosine kinase that is approved as therapy for patients with non–small cell lung cancer and advanced-stage pancreatic cancer.[1–4] It has been associated with anterior uveitis in 2 prior cases.[5,6] We report a 77-year-old female with stage IV squamous cell cancer of the lung with liver metastases who was treated with erlotinib starting 6 weeks before the onset of bilateral anterior uveitis. The uveitis was recalcitrant to topical corticosteroid therapy alone. Because she had not responded positively to previous systemic chemotherapy, the patient refused to stop erlotinib. The intraocular inflammation was successfully treated with a combination of topical and periocular steroids without drug cessation.

A 77-year-old presented with bilateral floaters for 1 week. She had an ocular history of a rhegmatogenous retinal detachment OD repaired with a scleral buckle and Fuchs dystrophy with a penetrating keratoplasty OU. She had no prior history of uveitis or any rheumatologic disease. Her medical history was significant for stage IV squamous cell cancer of the left lung with liver metastasis 8 months prior. She was treated with radiation to the lung followed by 4 cycles of carboplatin and gemcitabine, but demonstrated progression of her disease. Her chemotherapy was changed to oral erlotinib 150 mg daily approximately 6 weeks before presentation.

Her vision was 20/30 OD and 20/40 OS. Anterior segment showed multiple, nongranulomatous keratic precipitates OD, but none OS. There was 3+ anterior chamber (AC) cell with 1+ flare OD and 2+ cell with

1+ flare OS (Standardization of Uveitis Nomenclature). There was spillover of 2+ cell into the anterior vitreous OD and 1+ cell in the anterior vitreous OS. Fundus examination was otherwise unremarkable, except for stable drusen OU and a scleral buckle with laser scarring peripherally OD from the previous retinal detachment repair.

The patient was diagnosed with bilateral anterior uveitis. She was started on topical prednisolone acetate every 2 hours OD and 4 times daily OS. Three days later, there was no improvement in inflammation, and topical steroids were increased to every hour OD and every 2 hours OS. At this time, she also began to experience other systemic negative side effects of erlotinib, including diarrhea, rash, and dry skin, and the dose was decreased to 125 mg daily by her oncologist. One week later, neither her systemic negative side effects nor the inflammation OD were improved after the dose reduction. The patient did not want to stop the erlotinib, so she was treated with 40 mg posterior sub-Tenon's triamcinolone injection OD; 10 days after periocular steroid injection OD, the AC inflammation was improved with near resolution. Over the next 3 months, the inflammation slowly resolved and the prednisolone was slowly tapered to daily OU. Over the entire treatment period, the best corrected visual acuity remained stable. She remained on 125 mg erlotinib daily, but unfortunately brain metastasis developed and the patient died 3 months later.

Bilateral anterior uveitis associated with erlotinib has been reported in only 2 prior cases,[5,6] both of which required drug cessation for resolution of uveitis. We show that erlotinib may be continued and AC inflammation successfully treated with periocular corticosteroids. This is particularly useful in patients who prefer not to stop the medication because they have previously not responded to other chemotherapeutic regimens, as in our case. This case is unique in that posterior sub-Tenon's triamcinolone was required to achieve optimal control of the anterior uveitis.

It has been reported that erlotinib may also cause conjunctivitis, keratoconjunctivitis sicca, trichomegaly, and corneal epithelial defects in addition to anterior uveitis.[7,8] We recommend that patients taking erlotinib be referred to an ophthalmologist should any uveitis develop. Those who experience uveitis should discuss the risks and benefits of continued use with their oncologist and ophthalmologist in light of the possible successful treatment with periocular corticosteroids.

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Secondary localized corneal amyloidosis caused by lower eyelid epiblepharon

Localized corneal amyloidosis is a condition in which the protein amyloid is deposited in the cornea. Secondary corneal amyloidosis (SCA) is rare and usually develops after chronic corneal disorder or inflammation, such as keratoconus, trachoma, and keratitis.[1,2] SCA could also be caused by long-lasting touching of cilia in patients with trichiasis, and only about 30 cases have been reported so far.[3–5] To our knowledge, SCA has not been previously reported in patients with epiblepharon. In this article, we report a patient with epiblepharon who experienced development of SCA.

A 15-year-old female visited our clinic reporting a white lesion on her left cornea that developed 2 years prior. Her best corrected visual acuity was 20/20 OD and 20/40 OS. On her left cornea, a white amorphous elevated lesion 3 mm in diameter was observed just below the pupil centre (Fig. 1A, 1B). She also had left lower eyelid epiblepharon, which has been present since childhood, and cilia were touching and pricking her cornea right at the white lesion. There was no epiblepharon in her upper eyelids and right lower eyelid.