progressed rapidly. CBZ is used to treat manic depressive disorders and epilepsy. The reported systemic side effects of CBZ are rash, bone marrow suppression, thrombocytopenia, and liver and renal dysfunction, but none of these was found in our patient. The visual side effects are disorders of the extraocular muscles, including diplopia, nystagmus and accommodative palsy, visual hallucinations, pigmented retinopathy, and papilledema.1 Punctate cortical lens opacity has also been reported,1,3 but there has been no report of this in Japan. Lens opacities due to CBZ and sodium phenobarbital occasionally include punctate opacities in the cortex. The mechanism of development of cataracts caused by these drugs is not clear.

Our patient had received CBZ for 3 years with a total dose of 248g, and phenobarbital for 8 years with a total dose of 153g. He had no punctate opacities. However, these large doses of the drugs might be related to the occurrence of cataracts. His head injury might not have been directly related to the bilateral cataracts, because the right eyeball itself was not hit and the cataracts were bilateral. He did have a very slight acute subdural hemorrhage on the right side after being hit in the head by an iron ball at school. Myotonic dystrophy and hypoparathyroidism can be ruled out on the basis of laboratory examination data, and he had no psychiatric disease. He had normal neurological signs, including muscle force. Bilaterality and rash progression seem to be characteristic of anticonvulsant-induced cataract. Rash progression is hard to explain, but in our patient the change of the anticonvulsant and his head injury might be clues.

We successfully treated this young patient with acute anterior and posterior subcapsular cataracts induced by anticonvulsants, and he eventually regained normal vision.
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Impairment of central visual function due to macular degeneration, a deep keratoplasty (DKLP) was performed on the left eye.

One month after DKLP, his visual acuity OS improved to 0.03 (noncorrectable) with a clear graft, and his quality of life was significantly improved because he was now able to walk by himself, thanks to the improved peripheral vision (Fig. 2). The visual acuity in the right eye remained at finger counting at 30cm even 1 year after the macular hemorrhage; however, we are planning a DKLP on the right eye for better quality of vision.

A homozygous single base-pair transition (ACG to ATG, tyrosine to methionine) in codon 321, exon 6, was detected in the LCAT gene in both the patient and his younger brother. The parents were first cousins whose families were originally from Nakatsugawa, Japan.

Comment

Horven and Gjone reported ruptures of Bruch’s membrane that resembled angioid streaks as an ocular complication of familial LCAT deficiency. The macular degeneration and hemorrhages observed in our patient should be considered complications secondary to the alterations in Bruch’s membrane, thus confirming the suggestion of these earlier authors. From our patient’s history, it was highly likely that he already had macular degeneration bilaterally and that the latent submacular neovascularization led to the sudden loss of vision after the macular hemorrhage in the right eye.

Keratoplasty is rarely required in patients with LCAT deficiency because the opacification of the cornea progresses slowly, and visual acuity is only slightly reduced by the corneal haziness. The main cause of the bilateral decreased visual acuity of our patient at the first visit was probably the macular degeneration instead of the corneal opacity. The visual acuity, which was finger counting at 30cm before surgery, improved slightly to 0.03 (noncorrectable) even after DKLP. However, a remarkable change in the quality of life was attained owing to the improvement of peripheral vision. Because the diffuse haziness of the corneal stroma in eyes with LCAT deficiency is accentuated

Figure 1. In a 66-year-old patient with familial lecithin: cholesterol acyltransferase deficiency, diffuse, punctate corneal opacities can be seen bilaterally in association with peripheral ring opacity.

Figure 2. Fundus photograph of left eye of patient after deep lamellar keratoplasty showing ruptures of Bruch’s membrane (arrowheads). The dull reflex of the macular area in the left eye suggested a history of macular degeneration.

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