**Sir,
 A woman with bilateral maculopathy and acquired
 vitamin B12 deficiency**

Although retinopathy has been described in patients with congenital methylmalonic aciduria (MMA)¹ and

Fanconi anemia,² to our knowledge, there have been no reported cases of maculopathy in patients with acquired vitamin B12 deficiency. The prevalence of vitamin B12 deficiency is ~15% in the elderly,³ a population also affected by age-related macular degeneration.

Figure 1 Brain and spinal cord magnetic resonance imaging (MRI) and ophthalmologic diagnostic tests. MRI of the brain and spinal cord revealed T2-weighted hyperintensity (arrowheads) of the entire dorsal columns from the skull base to the conus (a). Color fundus photograph of the right and left eyes (b and c) showing round well-circumscribed subfoveal areas of RPE atrophy with numerous small (50–100 μm) flat yellowish deposits limited to the area of atrophy in both maculae. Fluorescein angiogram demonstrating early hyperfluorescence and late staining in the macula of the right and left eyes (d and e). Spectral domain optical coherence tomography (SD-OCT) imaging revealed normal retinal nerve fiber layers of the right and left eyes at presentation (f) and 6 weeks (g) after vitamin B12 supplementation. Superimposed retinal nerve fiber layer thickness for the right and left eyes at presentation and 6 weeks after treatment (h). SD-OCT of the right (i) and left (j) maculae at presentation showing focal disruptions of the inner segment ellipsoid and RPE layers without associated subretinal fluid or intraretinal fluid (central macular thickness was 255 μm for the right eye and 233 μm for the left eye). Repeat fundus photos and SD-OCT images of the right and left maculae after treatment were unchanged (photos not shown).

Case report

A 62-year-old Caucasian woman with a 12-month history of progressive paresthesias below the first lumbar level (L1) was hospitalized for rapid onset of ascending sensory impairment to the sixth thoracic level (T6), ataxia, saddle anesthesia, and diminishing central vision. Best-corrected visual acuity (BCVA) was 20/40 in the right eye and 20/400 in the left eye. Her BCVA had been 20/25 bilaterally two years prior. Color discrimination was severely diminished at 4/12 Ishihara color plates in the right and 1/12 in the left. Dilated fundus exams showed small flat yellowish deposits limited to well-circumscribed subfoveal areas of retinal pigment epithelium (RPE) atrophy (Figure 1). There was no optic nerve pallor. The fundus appearance and fluorescein angiogram findings were similar to those described in cases of congenital MMA.^{4,5} Full-field electroretinograms revealed mildly reduced scotopic and photopic responses in the left eye.

Despite a low normal level of vitamin B12 (298 pg/ml; reference 180–914 pg/ml), the patient's serum methylmalonic acid (0.30 μ M/l; reference <0.30 μ M/l) and homocysteine (22.2 μ M/l; reference 6.2–15.0 μ M/l) levels were elevated, consistent with a diagnosis of vitamin B12 deficiency. Furthermore, brain and spinal cord magnetic resonance imaging revealed T2-weighted hyperintensity of the entire dorsal columns, characteristic of vitamin B12 deficiency.

The patient was treated with cyanocobalamin injections by the inpatient Neurology service. Six weeks after treatment, her ataxia, paresthesias, as well as bowel and bladder function improved. Color discrimination improved to 8/12 in the right and 5/12 in the left eye. Nine months after treatment, her BCVA was stable at 20/40 in the right but improved from 20/400 to 20/40 in the left.

Comment

Vitamin B12 deficiency leads to low cellular levels of methionine and elevated levels of homocysteine. These alterations have either been hypothesized or shown to disrupt RPE, photoreceptor, and ganglion cell function.^{1,5,6} The partial reversibility of the patient's visual function may reflect the normalization of her homocysteine levels after treatment. This case suggests that vitamin B12 deficiency should be considered in the differential diagnosis of elderly patients presenting with worsening vision and macular findings that might otherwise be mistaken for new or worsening AMD. These patients may benefit from vitamin B12 screening as early detection and treatment may alter disease progression.

Conflict of interest

The authors declare no conflict of interest.

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Sir,

A case of accidental retinal injury by cosmetic laser

With the usage of laser in the fields of industry, research, and military, the risk of potential injuries is very real. We would like to report a patient who suffered a cosmetic laser-induced retinal injury.

Case report

A 25-year-old female was accidentally exposed to a cosmetic laser (SPECTRA-VRM II, MAX Engineering Ltd, Goyang City, South Korea) in 2012. The laser fired inadvertently when the patient was adjusting target without the use of her protective goggles. Immediately after the exposure, she experienced a bright flash of light followed by floaters in front of her right eye. The pulse duration and energy of the laser were not assessed. The patient's best-corrected visual acuity was 20/20 OD and 20/20 OS. Intraocular pressure and anterior segment biomicroscopy were unremarkable. Fundoscopic examination of her right eye revealed an inferior retinal laser burn and vitreous hemorrhage (Figure 1a). Prednisolone and vitamins were administered orally. Three days later the floaters in front of her right eye decreased, and fundoscopic examination of the eye revealed a laser burn with retina edema around it and vitreous hemorrhage (Figure 1b). Unfortunately, the patient did not return for further follow-up.

Comment

Some laser-induced eye injuries have been reported in the past. However, there has not been a case of cosmetic laser-induced retinal injury reported before.