preserved posterior pole were detected in both eyes of our patient as if the axial growth and subsequent staphylomatous bowing mainly affected the mideye. This would also explain the absence of significant chorioretinal atrophy in the fundus area.

Measurements of the ocular transverse diameters by ultrasonic biometry were 25 mm bilaterally in agreement with the mean value of 25.5 mm obtained on enucleated eyes harboring lacquer cracks. In consideration of the young age of our patient (18 years) and his relatively low refractive error in the affected eye (−6.50 D sphere), lacquer cracks could represent an initial finding before becoming incorporated into a larger area of myopic chorioretinal atrophy that develops with age. Indeed, transition to other myopic fundus changes have been demonstrated in 36.1% of 66 eyes, these being mainly represented by patchy or diffuse chorioretinal atrophy. Patients showing progression to patchy atrophy were younger and had longer axial lengths than those who demonstrated diffuse atrophy eyes. In the present case, the lesion appeared to be stable 1 year following initial examination.

Lacquer cracks must be differentiated from 2 similar diseases of Bruch membrane, namely angioid streaks and choroidal ruptures. Angioid streaks usually emanate from the disc, tend to be straighter, and are reddish in color. Choroidal ruptures, on the other hand, share with lacquer cracks similar distribution, color, and fluorescein angiographic appearance but are caused by a noticeable traumatic event. Traumatic choroidal ruptures may form arc-shaped patterns at various distances out from the optic nerve with the optic head at the center of the curves. However, they appear to be somewhat thicker and do not criss-cross.

In doubtful cases, fluorescein angiography can help in clarifying the clinical diagnosis and should include views of the midperiphery. Instrumental and clinical findings in our patient were in agreement with lacquer cracks. Recent cases of lacquer crack development following photodynamic therapy or laser treatment of choroidal neovascularization have been reported. A causal relationship between laser photocoagulation and lesion formation was suggested. Vigorous eye rubbing has also been advocated as a possible predisposing factor to lacquer crack formation. Therefore, a complementary role for traumatic events should be taken into consideration in the pathogenesis of lacquer cracks, and these may have precipitated an impending Bruch membrane rupture in our patient.

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Retinopathy and Choroidopathy as the Initial Signs of Hypertensive Brainstem Encephalopathy

In hypertensive encephalopathy, brain magnetic resonance imaging characteristically shows a posterior leuкоencephalopathy that predominantly affects the white matter of the parieto-occipital regions. Brainstem hypertensive encephalopathy predominantly affects the brainstem and cerebellum while sparing the parieto-occipital region and has not been documented in the ophthalmologic literature.

Report of a Case. A 46-year-old white woman described intermittent nausea, daily headaches, and a central scotoma in the right eye for approximately 1 week following blunt trauma to the face. Visual acuity was 20/200 OD and 20/40 OS. Pupils were brisk without an afferent pupillary defect. The anterior segments and intraocular pressures were normal. Fundoscopy of the right eye revealed marked optic disc edema and diffuse intraretinal exudate involving the fovea (Figure 1). The retinal vessels were tortuous with marked arteriolar constriction. Numerous areas of linear pigment epithelial hypertrophy with surrounding hypopigmentation were seen throughout the periphery of both fundi. The left optic disc was swollen to a lesser degree. Goldman perimetry revealed a large cecocentral scotoma in the right eye and an enlarged blind spot in the left eye. Blood pressure was 250/145 mm Hg. She was referred to the emergency department.

The patient was admitted to the neurological intensive care unit where her blood pressure was controlled using intravenous nicardipine along with metoprolol, amlopidine, and hydrochlorothiazide. Neurologic examination findings remained unchanged, and testing for causes of secondary hypertension was unrevealing. Magnetic resonance imaging revealed abnormal signal and symmetric enlargement of the pons and, to a lesser degree, the medulla and midbrain, extending into the middle cerebellar peduncles and the adjacent deep cerebellum (Figure 2A). The cerebellar tonsils were displaced downward through the foramen magnum. Early hydrocephalus was suggested by dilation of the lateral and third ventricles.

Repeat brain magnetic resonance imaging 8 days later showed marked improvement in the appearance of the entire brainstem with much less crowding of the foramen magnum (Figure 2B). On the day of hospital discharge, she was receiving oral valsartan, metoprolol, amlopidine, hydrochlorothiazide, and potassium and had a blood pressure of 133/69 mm Hg. Fourteen days after her initial examination,
Funduscopy revealed markedly improved disc edema with persistent macular exudates and stable vision in both eyes.

**Comment.** Despite the presence of extensive brainstem lesions on neuroimaging of brainstem hypertensive encephalopathy, there are often few symptoms other than headache, and this has been coined “clinical radiologic dissociation.”

The differential diagnosis of the neuroimaging of hypertensive brainstem encephalopathy includes neoplasm of the brainstem and infectious encephalitis. The lack of cranial nerve findings and other focal deficits on neurological examination along with the rapid clinical evolution and resolution of symptoms with correction of hypertension helps establish the diagnosis of hypertensive encephalopathy. Neuroimaging typically reveals complete resolution of brainstem findings following blood pressure control. Abnormal vision on initial examination occurred in one third of published cases. Severe hypertensive retinopathy, disc edema, optic nerve pallor, ptosis, and ophthalmoplegia have been documented in these patients. Our patient illustrates that the ophthalmologist might be the first to diagnose hypertensive brainstem encephalopathy because of decreased vision from retinopathy and chorioidopathy.

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Lesions affecting the dorsal midbrain can result in a constellation of ocular findings such as vertical gaze disturbances, convergence retraction nystagmus, light-near dissociation of pupils, and eyelid retraction.1—2 Although bilateral superior oblique palsy can occur after a stroke, its occurrence secondary to nontraumatic brainstem hemorrhage is extremely rare.1 We report a combination of dorsal midbrain syndrome and bilateral superior oblique palsy following brainstem hemorrhage.

Report of a Case. A 43-year-old man noted sudden onset of binocular vertical and torsional diplopia subsequent to a stroke 2 years prior to presentation. He complained of oscillopsia more pronounced in upgaze along with an anomalous chin-down position since the stroke. He was receiving anticoagulant therapy for coagulopathy at the time of the stroke and underwent a right frontal ventriculoperitoneal shunt for acute hydrocephalus secondary to the intracranial bleed.

Uncorrected visual acuity was 20/25 OU. Pupils were 3 mm in both eyes with a light-near dissociation. A 20° chin-down position was noted in primary gaze. Motility examination showed 30 prism diopter (Δ) esotropia with 4 Δ right hypertropia in primary position, 20 Δ esotropia with 7 Δ right hypertropia in upgaze, and 35 Δ esotropia with 4 Δ right hypertropia in downgaze. In right gaze, there was 20 Δ esotropia with 9 Δ left hypertropia, and in left gaze, there was 20 Δ esotropia with 10 Δ right hypertropia. Right head tilt revealed 20 Δ esotropia with 12 Δ left hypertropia, and left head tilt showed 25 Δ esotropia with 4 Δ left hypertropia. There was reduced depression in adduction and reduced elevation in both abduction and adduction in both eyes. Double Maddox rod test showed 30° excyclotorsion OS in primary gaze increasing to 42° in downgaze. An eyelid retraction and convergence retraction nystagmus was noted in attempted upgaze with hypometric vertical saccades both in upgaze and downgaze (Figure 1).

Sagittal noncontrast magnetic resonance imaging (Figure 2A) performed 3 months prior to ocular examination revealed prior brainstem hemorrhage extending.