eral ptosis can see well in the nonptotic eye and do not have the stimulation to lift the ptotic eyelid.3

However, the influence of compensatory contraction of the levator–lower eyelid retractor may be limited in congenital ptosis because of improper or faulty development of the levator muscle.4 Another possible mechanism is the mechanical effect of upper eyelid lift during ptosis surgery. Upper eyelid lift can lead to lower eyelid elevation due to the circumferential structure of the orbicularis muscle and changeability of the canthi position.5

These 2 mechanisms may be operative at the same time. Compensatory contraction may be stronger in patients with poorer levator function, and the larger amount of intraoperative upper eyelid lift may result in greater elevation of the lower eyelid.

In conclusion, this study shows lower eyelid elevation after surgical correction of congenital ptosis, especially after frontalis suspension or in bilateral ptosis. Surgeons should inform patients that lower eyelids can displace upward after ptosis surgery and that preoperative lower scleral show can be diminished postoperatively.

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Comment. Preeclampsia usually has onset after the 20th week of pregnancy and is characterized by blood pressure higher than 140/90 mm Hg and a urine protein level of 300 mg/24 hours. It has been associated with varied visual disturbances and transient and permanent vision loss. Pathological findings related to vision loss include retinal vessel spasm and occlusion, choroidal infarction or choroidal effusions leading to serous retinal detachment, and focal edema and hemorrhages in the occipital cortex.1 In addition, optic disc edema may arise due to intracranial hypertension, systemic hypertension, or anterior ischemic optic neuropathy.

Visio loss due to optic neuropathy in the immediate postpartum period can have a variety of causes, including blood loss1 and anesthetic complications.2 We describe a 35-year-old woman with resolving preeclampsia who had sudden unilateral vision loss after giving birth and had a clinical presentation consistent with nonarteritic anterior ischemic optic neuropathy (NAION).

Report of a Case. A 35-year-old postpartum woman had sudden loss of vision in her right eye, noted approximately 8 days after vaginal delivery of her full-term baby. The patient had a history of preeclampsia (hypertension, protein in urine 452 mg/24 hours, and leg swelling) diagnosed 2 weeks before delivery. She had continued problems with blood pressure control since delivery, and she also reported severe headache prior to vision loss. There was no history of hypertension, smoking, or medication use. There was no significant ocular history. There was a history of gestational diabetes mellitus for the last 2 months of pregnancy. On initial examination, her blood pressure was 130/82 mm Hg and her weight was 95 kg. Her visual acuity was 20/200 OD and 20/15 OS. There was a relative afferent pupillary defect in the right eye. Ophthalmoscopy revealed superior segmental disc edema with some early pallor in the right eye and a normal disc (with a small cup-disc ratio) in the left eye (Figure 1). Results of the remainder of her ocular examination were normal. Findings on computed tomography and magnetic resonance imaging of the brain and orbits were normal. Results of the laboratory workup including complete blood cell count, erythrocyte sedimentation rate, and C-reactive protein level were normal and she was negative for antinuclear antibodies. Humphrey automated visual field testing (30-2 Sita Fast) demonstrated an inferior altitudinal defect in the right eye and a normal field in the left eye (Figure 1). The working diagnosis was NAION. On follow-up approximately 6 weeks after her initial visit, visual acuity was 20/30 OD and 20/20 OS. Findings of the remainder of the examination were stable except for the development of some segmental superior disc pallor in the right eye (Figure 2). Results of repeated automated visual field testing were also unchanged (Figure 2).

Figure 1. Disc photographs of the right (A) and left (B) eyes at the initial visit with corresponding 30-2 Humphrey visual field.

Figure 2. Disc photographs of the right (A) and left (B) eyes at follow-up with corresponding 30-2 Humphrey visual field.
Two instances of ischemic optic neuropathy associated with preeclampsia have been reported. However, in contrast to our case, both of these events began before delivery and involved bilateral disc edema and vision loss. Our case appears to be unique in that the symptoms appeared after delivery and the vision loss and disc edema were unilateral. The disc swelling in this case was sectoral and the visual field loss was altitudinal, characteristic of a typical presentation of NAION.

The pathophysiology of preeclampsia is believed to originate from the placenta. Cytotrophoblast cells in the embryo enter the uterine wall and invade the maternal uterine spiral arteries. These cells change from an epithelial phenotype to an endothelial phenotype. This vascular remodeling seems to be disrupted in preeclampsia, resulting in the production of high levels of antiangiogenic factors that enter the maternal circulation; these antiangiogenic factors disrupt the maternal endothelium, resulting in hypertension. The exact pathophysiology for NAION in preeclampsia remains elusive, but it is suggested that the uncontrolled hypertension leads to vasoconstriction or ischemia in the posterior ciliary artery circulation.

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COMMENTS AND OPINIONS

Recurrence of Retinopathy of Prematurity Following Bevacizumab Monotherapy: Is It Only the Tip of the Iceberg?

In 2011, a landmark article was published that could change the way retinopathy of prematurity (ROP) is treated. The Bevacizumab Eliminates the Angiogenic Threat of Retinopathy of Prematurity (BEAT-ROP) trial concluded that intravitreal injection of bevacizumab, 0.625 mg in 0.025 mL, had a beneficial effect compared with laser photocoagulation for zone 1, stage 3+ ROP. However, their end point for recurrence of disease was only 54 weeks’ postmenstrual age (PMA). Previously, Moshfeghi and Berrocal estimated that 47.7% of recurrences would have occurred after the 54 weeks’ PMA reported in the BEAT-ROP study as the primary end point for treatment success. In this journal, Hu et al described their clinical experience with recurrence of ROP following intravitreal injection of bevacizumab, adding further evidence that 54 weeks’ PMA is not a sufficient end point for observation.

Hu and colleagues reported 17 eyes of 9 patients who had recurrence of ROP requiring treatment on average 14.4 weeks after bevacizumab injection (range, 4-35 weeks). Notably, 5 eyes that progressed to having retinal detachment (RD) did so at a median age of 55 weeks’ PMA (range, 49-69 weeks). They mentioned the shortfalls of their study as retrospective, uncontrolled, and small in number. Also, the period in which their patients were seen in relation to recent publications on bevacizumab was not described, and patients were seen and managed by different ophthalmologists without prior examination by authors of the study in some cases. Thus, this retrospective study raises concerns about nonstandardized use of a new treatment modality. Most importantly, 2 patients had repeated intravitreal injections of bevacizumab within a relatively short period. One patient (patient 3) had 6 doses of bevacizumab, 0.625 mg (total, 3.75 mg), in a 7-week period, and another patient (patient 9) had 5 doses in the same period (total, 3.125 mg). Sato et al showed that a total dose injection of only 0.5 mg of bevacizumab causes a 6-fold reduction in serum vascular endothelial growth factor 2 weeks after injection in neonates. There are no data for repeated injections, but by implication, patients 3 and 9 in the article by Hu and colleagues were likely to have been subjected to even lower serum vascular endothelial growth factor levels. Systemic adverse effects remain the biggest concern among ROP experts in both the short and long term. We therefore welcome the recommendation that recurrences of ROP should be treated with laser, not just because of a reduced RD rate but also for protecting the patient from systemic complications.

We are in uncharted waters with regard to patterns of recurrence following bevacizumab treatment. Hu and colleagues defined recurrence as “arrest of anterior progression of retinal vasculature associated with a new demarcation line, ridge, or extraretinal fibrovascular proliferation (EFP) or leakage on fluorescein angiography, with or without recurrence of plus disease.” Recurrence did not require EFP. They also observed anatomical recurrence as anterior and/or posterior. This is different from the definitions in the BEAT-ROP study. There is a need for the larger ROP community to standardize the definition and re-treatment criteria for recurrence after bevacizumab injection. In the study by Hu and colleagues, both recurrence criteria and treatment were at the discretion of the treating ophthalmologist. Again, questions can be raised regarding some patients with the information provided in the article. For example, patient 9 was born at 32 weeks’ PMA, weighed 1843 g at birth, and reportedly developed aggressive posterior ROP in zone 1 with atypical stage 3 and plus disease at 35 weeks.