rine to more distal vessels may have led to vasoconstriction and subsequent vasospasm.

In conclusion, epinephrine can lead to OAO following accidental intra-arterial injection of subcutaneously administered local anesthetics. Hence, physicians should carefully administer local anesthesia while considering the possibility that such a complication may occur.

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Two Cases of Endogenous Endophthalmitis That Progressed to Globe Rupture

Dear Editor,

Infectious endophthalmitis is a condition in which the internal structures of the eye are invaded by replicating microorganisms. Early administration of antibiotics or early vitrectomy should be considered to preserve vision. However, the general health status including systemic infection should be considered in surgical intervention such as vitrectomy, especially in some cases of endogenous endophthalmitis. Whether vitrectomy is beneficial for endogenous endophthalmitis remains controversial, especially when visual outcome is predicted to be hopeless. Here we report two cases of endogenous endophthalmitis without vitrectomy that progressed to globe rupture, which was not expected.

A 56-year-old male patient with history of diabetes mel-

litus presented with blurred vision of the right eye for 10 days. Abdominal and chest computed tomography showed an emphysematous prostatic abscess with multiple pulmonary lesions. Thrombocytopenia ($18 \times 10^3/\mu\text{L}$) derived from disseminated intravascular coagulation was detected. He was administered an intravenous injection of cefepime and meropenem. His visual acuity was hand motion. Moderate corneal edema and 3+ cells were found in the anterior chamber. Fundus was not visible. A B-scan ultrasound disclosed obvious thick vitritis. With the impression of endogenous endophthalmitis, intravitreal injection of ceftazidime and vancomycin was performed. Culture of the anterior chamber showed extended spectrum beta-lactamase (-) *Klebsiella pneumoniae*. However, 1 day after the intravitreal injection, his visual acuity was not improved, and there was no sign of recovery. Thus, vitrectomy was considered. However, it was abandoned because the risks derived from systematic factors were high, including poor general condition with systemic infection. Most importantly, his thrombocytopenia did not meet the criteria for minor surgery. Furthermore, his visual outcome was predict-

Fig. 1. (A) Photograph showing right proptosis with marked lid swelling. Conjunctiva shows severe chemosis and necrotic change; (B,C) computerized tomography scan of the orbit. Right globe with irregular contour shows nasal superior, posterior scleral breakthrough (red arrow), suggesting a ruptured globe. (D) Photograph showing right proptosis. Conjunctiva shows severe chemosis and conjunctival hemorrhage; (E,F) magnetic resonance imaging scans with contrast enhancement showing proptosis and anteroposterior elongation of the right globe with an irregular contour. A defect at the superolateral aspect (red arrow) is visible.

ed to be hopeless due to prolonged disease duration. However, after 3 days, proptosis became profound. The ipsilateral eyelids were diffusely stretched and retracted (Fig. 1A), suggesting eyeball rupture, which was unexpected. Orbital computed tomography revealed eyeball rupture (Fig. 1B and 1C). Evisceration with wide debridement was performed.

An 83-year-old male patient with history of hepatocellular carcinoma presented with blurred vision of the right eye for 1 week. His visual acuity of the right eye was hand motion. The ipsilateral eyelid was mildly swollen. Severe corneal edema and 4+ cells were found in the anterior chamber. A B-scan ultrasound disclosed vitritis. There was no history of ocular surgery or trauma. Endogenous endophthalmitis was our impression. Therefore, we performed intravitreal injection of ceftazidime and vancomycin with intravenous injection of ceftazidime and vancomycin. Culture of the the anterior chamber showed extended spectrum beta-lactamase (-) *Klebsiella pneumoniae*. One day after the intravitreal injection, his ocular state was stationary. However, due to thrombocytopenia ($36 \times 10^3/\mu\text{L}$) and his poor general condition, vitrectomy was not considered. The patient accepted the possibility of visual loss. However, proptosis and right eyelid swelling developed on the same day (Fig. 1D). Orbital magnetic resonance imaging revealed eyeball rupture (Fig. 1E and 1F), for which evisceration was performed.

Vitrectomy has been accepted as a treatment option supplementary to intravitreal antimicrobial therapy in patients with moderate or severe infectious endophthalmitis [1]. However, in clinical practice, whether vitrectomy is beneficial for patients with poor general condition remains con-

troversial, especially when visual outcome is predicted to be hopeless. Vitrectomy was abandoned in our cases because the prognosis was predicted to be poor due to seriousness and prolonged disease period of endogenous endophthalmitis. Moreover, the two patients were in a poor general condition with thrombocytopenia. However, endophthalmitis progressed to orbital rupture. One systematic review with a total of 342 cases of endogenous bacterial endophthalmitis has reported that only five of 56 eyes (9%) undergoing vitrectomy required evisceration or enucleation, compared to 29 of 110 eyes (26%) that did not undergo vitrectomy ($p = 0.08$) [2]. These reports and our cases showed that prompt vitrectomy should be considered in endophthalmitis, even if visual prognosis does not seem to be good.

In conclusion, early vitrectomy should be considered in endogenous endophthalmitis to save the eyeball even if visual outcome is predicted to be hopeless. Even if vitrectomy is not feasible due to systematic factors, the patient should be informed beforehand that endophthalmitis without vitrectomy can lead to not only blindness, but also evisceration. Prompt surgical treatment can save the eyeball and prevent the spread of infection to adjacent tissues of these patients.

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Serial Photographic Monitoring of Spontaneous Clearance of Corneal Blood Stain in a Child with Traumatic Hyphema

Dear Editor,

Corneal blood staining can occur as a complication of traumatic hyphema with frequencies of 2% to 11%, particularly in the conditions of total hyphema, rebleeding, high intraocular pressure (IOP), and endothelial dysfunction [1,2]. Surgical interventions are recommended in eyes with high IOP and sustained blood in the anterior chamber to decrease the incidence of complications [3,4]. We describe the natural history of corneal blood staining following traumatic hyphema in an 8-year-old boy by presenting serial photography of the spontaneous clearance process.

An 8-year-old boy visited Seoul St. Mary's Hospital with decreased visual acuity (VA) and corneal opacity after blunt trauma to the right eye 30 days prior. He had undergone irrigation of the anterior chamber for increased IOP combined with hyphema occupying over 90% of the anterior chamber at another hospital on the 6th day after trauma. There were no descriptions of cataract formation at that point. VA was reduced to hand-motion and IOP was 8 mmHg at the initial visit to our hospital (1-month post-trauma). Slit lamp examination revealed rust-colored corneal opacity occupying the entire cornea, which obscured the iris, lens, and posterior segment (Fig. 1A). The affected cornea was mildly edematous, and central corneal thickness was 703 μ m. Two months after the trauma, the blood-

stained area seemed to clear from the peripheral limbus (Fig. 1B) with slight improvement in VA, so we chose to follow-up regularly instead of performing any invasive intervention. At post-traumatic 3 months, the diameter of the blood-stained area had decreased and the patient was prescribed partial-patch of the left eye (Fig. 1C). Centripetal clearance had continued to improve at 6 months, with VA at 20 / 100 (Fig. 1D). He was prescribed myopic glasses with maintenance of occlusion therapy. VA was corrected to 20 / 40 with a 2.5 $\times$ 3.0-mm-sized central discoid lesion at 12 months (Fig. 1E). The fundus was clearly visible at that time. Complete corneal clearance was achieved, and he showed a final VA of 20 / 40 at 15 months (Fig. 1F). Traumatic mydriasis and cataract remained. Corneal endothelial cell density was 1,282 cells/mm². No stromal or endothelial opacities were detected in the affected eye.

Corneal blood staining usually occurs in the setting of large persistent hyphema, increased IOP, and corneal endothelial cell dysfunction [1,2]. Hemoglobin is released from erythrocytes into the anterior chamber. Increased IOP may aggravate stromal penetration of hemoglobin and its breakdown products through the endothelium whose barrier function has been anatomically and physiologically compromised. Hemoglobin and its breakdown products aggregate within the stromal lamellae and are phagocytized and metabolized by keratocytes, which then produce intracellular hemosiderin [1,2].

The corneal blood staining cleared without any permanent corneal opacities in the present 8-year-old boy even though the total clearing process lasted 15 months. Decreased endothelial cell density could be caused by either mechanical disruption of the endothelium or photosensitization of the endothelium by hemoglobin-derived porphyrins [5]. A VA of