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A Case of Ophthalmic Artery Occlusion Following Subcutaneous Injection of Epinephrine Mixed with Lidocaine into the Supratrochlear Area

Dear Editor,

Local anesthesia with an epinephrine-lidocaine mixture has been widely used in cosmetic facial procedures and oculoplastic surgery. Although the known side effects of local anesthesia include pain and hematoma in the injected area, we encountered a case of atypical ophthalmic artery occlusion (OAO) after subcutaneous injection of an epinephrine-lidocaine mixture into the supratrochlear area.

A 47-year-old woman complained of sudden visual disturbances in her right eye, which had developed 3 days earlier. The best-corrected visual acuity was 0.03, and a grade-II afferent pupillary defect was observed. Upon slit lamp examination, many red blood cells were found in the anterior chamber. Fundus examination revealed cotton wool patches (CWPs) of various sizes, and retinal hemorrhages at the posterior pole. Moreover, large preretinal hemorrhages were observed (Fig. 1A). Optical coherence tomography revealed hyperreflectivity in the inner retinal layer and multiple preretinal hemorrhages (Fig. 1B). Fluorescein angiography displayed slightly delayed chorioretinal filling, definite peripheral nonperfusion areas at the nasal and temporal periphery and vascular leakage and staining (Fig. 1C-1E).

The patient was relatively healthy before the episode. However, 3 days earlier, she had visited a local cosmetic clinic for facial filler injection and, before the procedure, received a subcutaneous injection containing 1 : 100,000 epinephrine and 2% lidocaine in the right supratrochlear area. She reported that it was the first injection in the procedure. Immediately after the injection, severe ocular pain, a thunder-like entoptic phenomenon, mydriasis, ptosis, and visual disturbances developed. She visited an emergency clinic and was diagnosed with central retinal artery occlusion (CRAO). Indocyanine green angiography revealed hypoperfusion of the choroidal arteries and choriocapillaris (Fig. 1F and 1G). Furthermore, optical coherence tomography angiography showed capillary loss in all retinal layers and in the choriocapillaris (Fig. 1H). We suspected that epinephrine had induced atypical OAO, and treated this patient with high-dose corticosteroid therapy for 3 days. Thereafter, the retinal hemorrhages increased in size and number and changed in shape (Fig. 1I). Four weeks after the initiation of treatment, most of the retinal hemorrhages and CWPs had been absorbed (Fig. 1J); however, count-fingers vision persisted and the visual field defect was not resolved (Fig. 1K and 1L).

Epinephrine acts peripherally on α-adrenergic receptors, resulting in the constriction of blood vessels [1]. Previous reports have described embolic occlusion caused by various materials, such as fillers or autologous fat. These materials can cause CRAO, OAO and/or cerebral infarction [2]. However, our particular patient had not received a filler injection, and her symptoms differed from the more devastating symptoms of filler-associated OAO, which include no recovery of light perception, visible filler material with arterial segmentation, and more complete chorioretinal artery filling defects on fluorescein angiography or indocyanine green angiography [3]. We suspect that other factors related to the epinephrine injection contributed to the development of this case. First, there were various changes in the clinical course of the disease, given that many of the initial signs of OAO had disappeared after 3 days. Furthermore, the CRAO-like fundus characteristics changed to retinal hemorrhages, CWPs, and peripheral nonperfusion areas. Although preretinal hemorrhage is not a characteristic finding of OAO, we think that the injected materials migrated in the anterograde direction and then induced peripheral vascular constriction and spasm, which contributed to peripheral nonperfusion, vascular leakage and preretinal hemorrhage caused by vessel wall damage. Second, epinephrine can cause visual disturbances including diplopia following trigeminal nerve block during dental procedures or following local anesthesia of the nasal mucosa during nasal surgery [4,5]. The proposed mechanism of transient OAO in these instances is vascular spasm resulting from intra-arterially injected epinephrine with retrograde migration. Finally, to the best of our knowledge, there have been no reported cases of OAO secondary to subcutaneous injection of local anesthetics alone. Thus, in our case, retrograde arterial displacement of the injected epinephrine from a branch of the supratrochlear artery into the ophthalmic arterial system may have blocked the ophthalmic artery immediately after injection. Through vasodilation over time, subsequent anterior movement of epineph

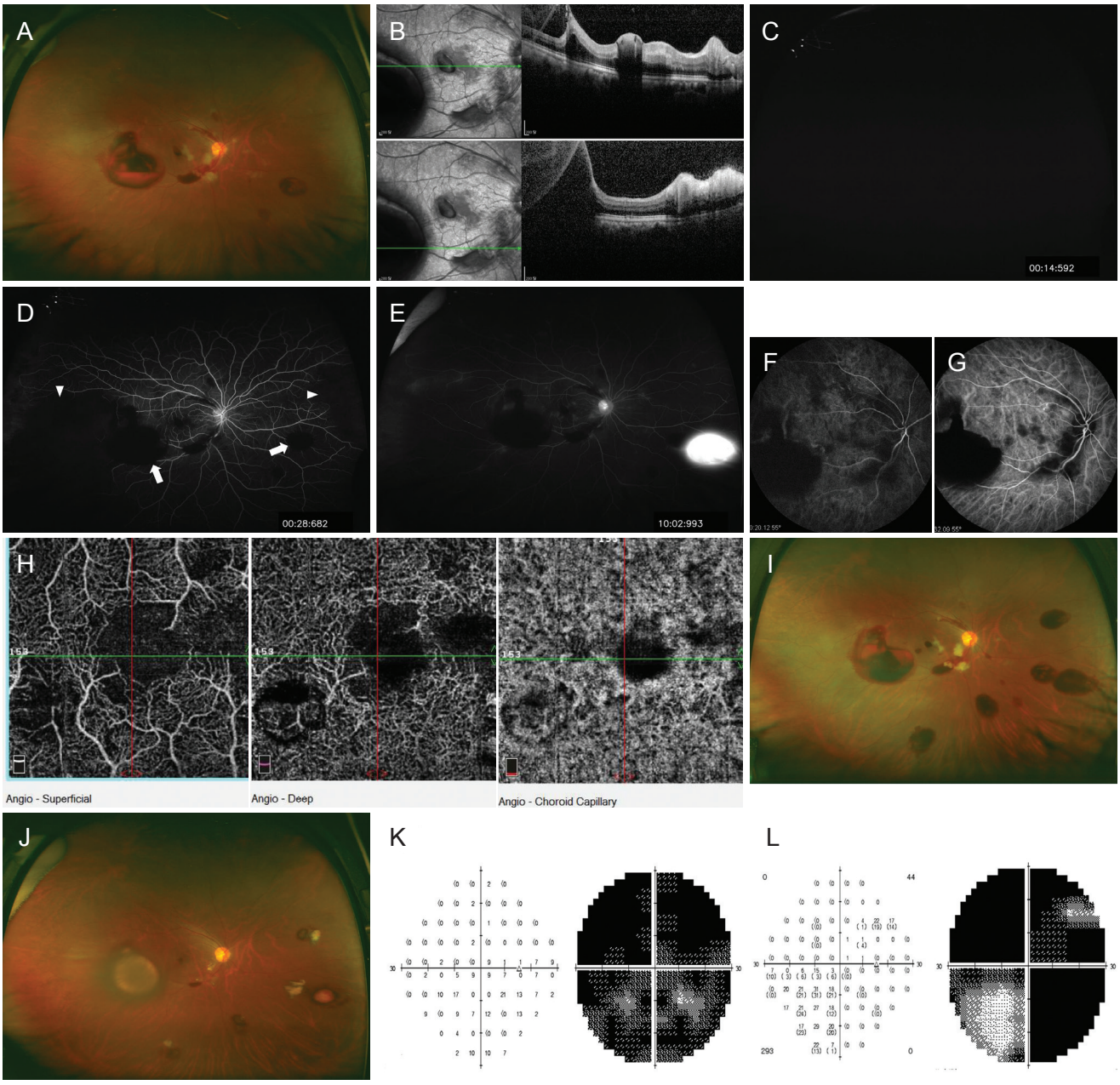

Fig. 1. (A) Wide fundus photograph of the right eye, 3 days after the initial presentation. Multiple cotton wool patches (CWPs) and retinal hemorrhages were observed around the optic nerve and macula. Large preretinal hemorrhages were observed at the nasal and temporal mid-peripheral retina. (B) Optical coherence tomography of the right eye, 3 days after the initial presentation. Preretinal hemorrhages of various sizes, posterior shadowing, and increased inner retinal hyperreflectivity (suggestive of ischemia of the inner retina) were observed. (C-E) Wide fluorescein angiography of the right eye, 3 days after the initial presentation. (C) A chorioretinal artery filling delay was seen in the early phase. (D) Multiple blocked fluorescent areas due to preretinal hemorrhages (arrows), and peripheral nonperfusion areas in the nasal and temporal retina (arrowheads) were noted in the arteriovenous phase. (E) Multiple vascular leakages and stains were observed in the late phase. (F,G) Indocyanine green angiography, 7 days after the initial presentation, revealed delayed filling of the choroidal artery in the early phase, and a segmented filling defect of the choriocapillaris. (H) Optical coherence tomography angiography (3 × 3 mm) exhibited disruption of the foveal avascular zone in the superficial and deep capillary plexuses, and multiple-capillary nonperfusion at the level of the superficial and deep retinal capillaries and choriocapillaris. (I) Immediately after the 3-day course of high-dose intravenous steroid therapy, wide fundus photography exhibited increased CWP sizes, retinal hemorrhages, and newly developed nasal and inferior retinal hemorrhages, compared with fundus findings at the first visit (A). (J) Four weeks after high-dose steroid treatment, most retinal hemorrhages and CWPs had disappeared. (K) Right eye 30-2 Humphrey visual field examination of the right eye, 3 days after initial presentation displaying extensive central, ceco-central and para-central scotomas. (L) Four weeks after high-dose steroid treatment, the visual field examination results revealed further deterioration; stimulus III could not be performed, and stimulus V displayed marked deterioration of the superonasal and temporal scotoma with inferonasal field sparing.

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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Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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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 mellitus 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 ($18 \times 10^3/\mu L$) 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-