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

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Introduction

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.

Case presentation

A 47-year-old woman complained of sudden visual disturbances in her right eye, which had developed 3 days earlier. The BCVA was 0.03, and a grade-II afferent pupillary defect was observed. Upon slit lamp examination, many RBCs were found in the anterior chamber. Fundus examination revealed multiple cotton wool patches (CWP), and retinal hemorrhages at the posterior pole, which affected the fovea. Moreover, large preretinal hemorrhages were observed (Fig. 1a). Optical coherence tomography (OCT) revealed hyperreflectivity in the inner retinal layer and multiple preretinal hemorrhages (Fig. 1b). Fluorescein angiography (FA) displayed slightly delayed chorioretinal filling, a definite peripheral nonperfusion area at the nasal and temporal periphery and late peripheral vascular leakage and staining (Fig. 1c-e).

We suspected that epinephrine had induced atypical OAO, and managed with high-dose corticosteroid therapy for 3 days. Thereafter, the retinal hemorrhages increased in size and number and changed in shape (Fig. 3a-b). Four weeks later, most of the retinal hemorrhages and CWP had been absorbed (Fig. 3c); however, count-fingers vision persisted and the visual field defect was not resolved (Fig. 4).

Discussion

Previous reports have described embolic occlusion caused by cosmetic fillers or autologous fat. However, our particular patient had not received a filler injection, and her symptoms differed from filler-associated OAO. We suspect that other factors related to the epinephrine injection contributed to the development of this case.

1) 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. And CRAO-like fundus findings changed to multiple retinal hemorrhages, CWP, and peripheral nonperfusion areas.

2) Epinephrine can cause transient OAO following trigeminal nerve block during dental procedures or local anesthesia of the nasal mucosa during nasal surgery. The proposed mechanism in those instances is vascular spasm resulting from intra-arterially injected epinephrine with retrograde migration.

3) There have been no reported cases of OAO secondary to subcutaneous injection of local anesthetics alone.

Epinephrine acts peripherally on α-adrenergic receptors, resulting in the constriction of blood vessels. 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 epinephrine to more distal vessels may have led to convascstruction and subsequent vasospasm.

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.

References