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Case Report

Ipsilateral, Multi-Phasic Retinal Vascular Events following Intralesional Triamcinolone Acetonide Injection for Earlobe Keloid: A Case Report

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Keywords

Keloid · Intralesional triamcinolone · Branch retinal artery occlusion · Central retinal vein occlusion · Case report

Abstract

Introduction: Intralesional triamcinolone acetonide is a widely used treatment for scarring skin conditions such as keloid and is known to have retinal vascular complications if administered in the periocular region. ***Case Presentation:*** A 32-year-old female experienced a prompt onset transient vision decrease and a delayed onset, slow-resolving vison loss in the right eye (OD) following the last of a series of triamcinolone acetonide corticosteroid (TAC) injections in her right earlobe for a disfiguring keloid. Clinically, she developed a branch retinal arterial occlusion accompanied by features of a central retinal vein occlusion. The TAC particles that entered the retinal circulation are implicated in the thrombo-embolic occlusion of the branch retinal artery and subsequent blockage of the central retinal vein. ***Conclusion:*** This case should alert clinicians that there is always a potential hazard for retinal vascular occlusion when corticosteroid is injected in the region of the head and face because of the rich anastomoses between the external and internal carotid artery circulations.

Introduction

Intralesional triamcinolone acetonide corticosteroid (TAC) injections have been a mainstay therapy for keloid due to its anti-inflammatory properties, inhibition of fibroblast proliferation, and other therapeutic mechanisms [1]. After more than half a century, the

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modern therapy of intralesional steroid injection has been modified in order to minimize systemic and local side effects [2, 3]. These modifications have consisted of different corticosteroid agents, injection dosing, injection site, and technique variation [2]. As a result, vision loss has rarely been reported with low pressure injections of TAC with 2.5 mg dosing around the eyes, except for cases of TAC injections to treat periorbital hemangiomas and intranasal lesions [3]. Since vision loss is rarely associated with this injection, the underlying mechanism of TAC-related occlusion of the retinal vasculature deserves scrutiny in every case [4]. Herein, we report a case of prompt and delayed onset vision loss, corresponding to a retinal arterial occlusion (RAO) with retinal venous involvement in the eye ipsilateral to the injected earlobe.

Case Report

A 32-year-old African-American woman with a history of keloid formation, uterine fibroids, and mild congenital lymphedema presented with 30 min of painless vision loss OD, described as "gray splotches in the vision that did not go away with blinking." One day prior to presentation, she received the last injection dose of 0.3 mL of 20 mg/mL intralesional TAC administered by a dermatologist to treat a large keloid of the right earlobe (shown in Fig. 1a). Of note, she had received at least eight injections in the past in the preceding 2 years without sequelae. Although her ophthalmic examination on that day of initial presentation was reported as unremarkable, review of fundus images from that visit revealed engorgement of the retinal venous circulation OD (shown in Fig. 1b) compared to that of the uninvolved left eye (OS; shown in Fig. 1c). OCT revealed no macular edema. Three weeks later, the patient experienced another 30-min episode with identical symptoms, which resolved without incident. However, 3 days following this transient episode, the patient represented to our service with a third, non-resolving episode of painless vision loss in OD of greater than 1 day duration.

On examination, her best corrected visual acuity was 20/400 OD and 20/20 OS, with no relative afferent pupillary defect. The intraocular pressures were normal. The anterior segment was unremarkable. Fundus OD revealed retinal whitening of the nasal macula adjacent to the nerve (shown in Fig. 2a). Compared to OS (shown in Fig. 2b), OD showed an edematous optic nerve, nasal macula retinal whitening, dilated and tortuous retinal veins, and scattered dot blot hemorrhages in both the nasal and temporal mid-peripheries suggestive of a central retinal vein occlusion. OCT showed inner retinal hyper-reflectivity of the nasal macula and numerous hyper-reflective foci at the level of the inner nuclear layer in the temporal macula indicative of ischemia (shown in Fig. 2c), with the fellow eye appearing normal (shown in Fig. 2d). Fluorescein angiography showed staining of optic disc vessels OD, highlighting the dilation and tortuosity of the retinal veins without evident non-perfusion (shown in Fig. 2e), as compared to the fellow eye (shown in Fig. 2f). More notably, optical coherence tomography angiography (OCTA) OD showed a distinct area of flow void in the nasal macula at the level of the deep vascular plexus (shown in Fig. 2g), corresponding to the area of retinal whitening on exam and inner retinal hyper-reflectivity detected by OCT. OCTA OS demonstrated no flow void in the deep vascular plexus (shown in Fig. 2h).

In light of the noncontributory personal and family history, further laboratory workup was performed: PT, PTT, protein C, homocysteine, and factor V, ANA, RF, CRP, FTA-Abs, sickle cell screen, SPEP, hemoglobin A1c, and lipid panel. All of the above were within normal limits. Echocardiogram detected no abnormalities. Additional neuro-imaging, including MRI/MRA of the head and orbits also were negative. Further intralesional TAC injections for the keloid were suspended indefinitely, with treatment limited to compression and topical therapy.

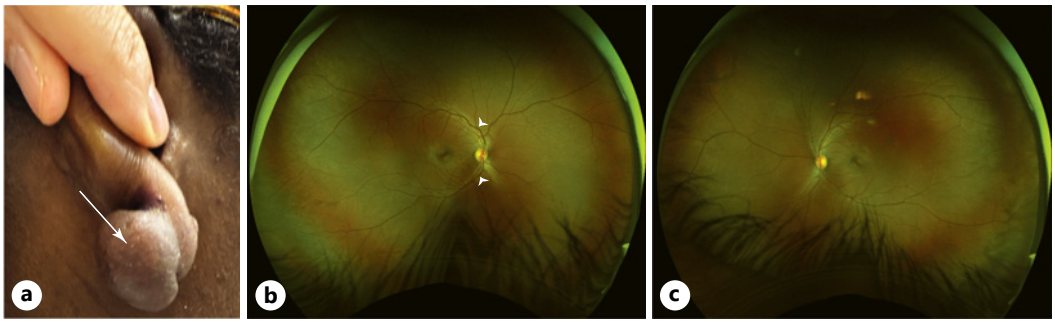

Fig. 1. a External photograph showing the large keloid (arrow) emanating from the patient's right earlobe on the day of the latest intralesional TAC injection. **b** Fundus photograph OD at the initial presentation showed only mildly dilated and engorged retinal veins (arrowheads). **c** Fundus photograph OS at the same visit appears normal.

Within 1 week, the visual symptoms OD subsided. By 6 months after presentation to our service, her best corrected visual acuity OD returned to 20/25 and the hemorrhages have resolved (shown in Fig. 3a). On OCT, the nasal macula OD has thinned substantially (shown in Fig. 3b). The patient has been monitored for recurrence and any signs of neovascular sequelae.

Discussion

The clinical impression of this case is that thromboembolic material, namely, TAC, introduced from local, superficial vessels entered the ophthalmic artery (OA) and eventually its downstream branching retinal artery, resulting in an iatrogenic branch retinal arterial occlusion (BRAO). The site of injection, the right earlobe, is supplied by the right posterior auricular artery, a branch of the external carotid artery [5]. The posterior auricular artery, a cutaneous artery, normally forms anastomoses that join collateral blood flow to regions of the face and head. These superficial arteries in the face, and their anatomic variations, can form communication between the internal and external carotid arteries. For example, OA, a direct branch from internal carotid artery, anastomosing with the external carotid artery system, is a possible point of entry for the embolic material that led to the eventual occlusion of the downstream branch retinal artery OD [6].

Intralesional TAC injection is a generally safe procedure widely used by dermatologists. However, a serious potential side effect of TAC injection, thromboembolism in the OA system, cannot be over-emphasized. The following are 5 aspects of our case that merit discussion. First, subcutaneous TAC introduced into the superficial arteries may gain access into the OA through various flow paths, including retrograde, anterograde, and various collaterals [6]. Second, based on previous studies, ~80% of undiluted TAC has particle sizes smaller than 20 μm, but approximate 3% of TAC particles have larger sizes (~500 μm) [7]. Relative to the retinal arteriolar diameter (~144 μm), the large TAC particle sizes confer high embolic risk to the retinal circulation. Packed particles within the medication can understandably lead to partial or complete vessel occlusion, especially in the crowded region of the optic nerve [8]. Other properties of TAC, including the propensity for particle aggregation and low solubility, have increased its hazard as a thromboembolic material [9]. Third, when TAC is injected into a blood vessel, serum factors may promote triamcinolone aggregation [9]. Fourth, in our case, a delayed onset of BRAO more than 3 weeks following TAC injections is notable and slightly unexpected. The proposed mechanism could be just a matter of time and buildup, by which

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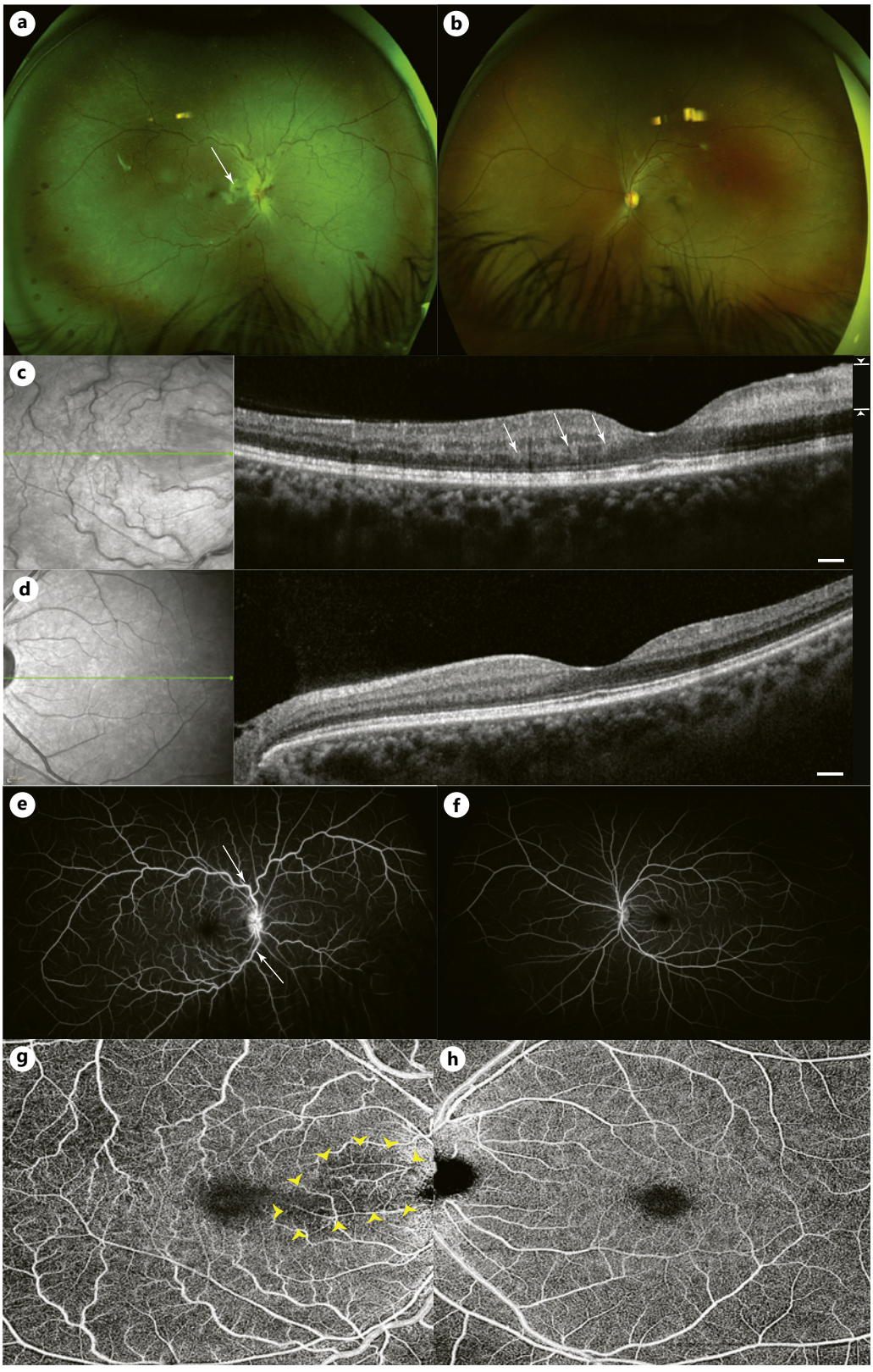

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accumulated TAC particles in the retinal circulation may occlude selected arterioles, causing a delayed onset RAO [10]. Fifth, although RAO's are classically characterized by retinal ischemic whitening and optic disc hyperemia/edema, the presence of peripheral retinal hemorrhages, venous dilatation, and vessel tortuosity suggests that the patient developed an accompanying retinal vein occlusion, as seen on the fundus (shown in Fig. 2a) and fluorescein angiography (shown in Fig. 2e). The associated RVO presumably resulted from venous thrombosis at the optic nerve level, proximal to the lamina cribrosa [11]. In a separate, but less likely scenario, progressive venous stasis precipitated by lodged TAC in the retinal venous circulation may have contributed to a sudden, complete blockage of the central retinal vein. Secondarily, a BRAO followed [11].

In summary, we have presented a case of intralesional TAC injection for an earlobe keloid that triggered an ipsilateral prompt and delayed BRAO. These arterial vascular events were both associated with venous congestion reminiscent of an RVO. This patient's experience has

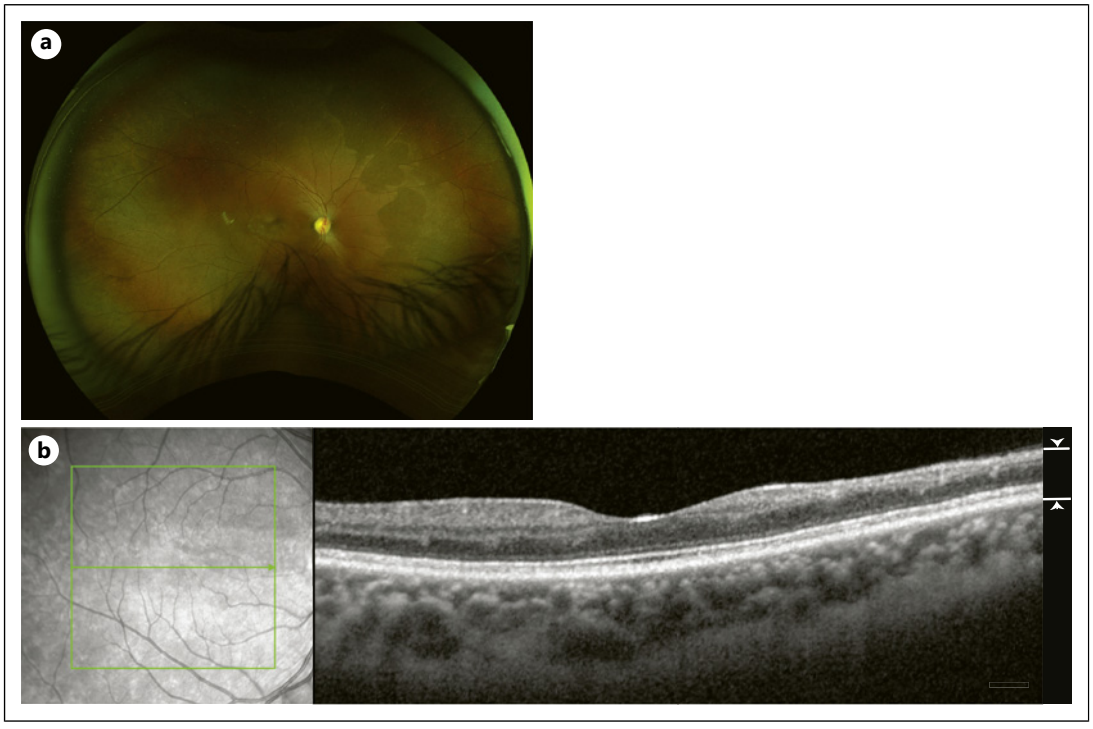

Fig. 3. a Fundus photograph OD at 6-month follow-up showed resolved hemorrhages. **b** OCT OD displayed nasal macular retinal thinning, with atrophy of the inner and middle layers (bracketed by arrowheads).

Fig. 2. a Three weeks later, fundus OD demonstrated optic nerve oedema, nasal macula retinal whitening (arrow), dilated and tortuous vessels, and 360° intraretinal hemorrhages. **b** Fundus OS remained normal. **c** OCT macula OD revealed intraretinal hyper-reflective foci in the inner nuclear and outer plexiform layers reminiscent of paracentral acute middle maculopathy (PAMM; arrows) lesions. The hyper-reflective inner retina nasal to the fovea is thickened (bracketed by arrowheads). **d** OCT macula OS was not thickened. The white bar at the bottom right of the OCTs is a scale for 200 μm. **e** FA OD in the later phases displayed staining of the disc, dilated veins (arrows), but without leakage or non-perfusion. **f** OS showed normal vessels and disc. **g** OCTA OD showed an evident flow void in the nasal macula (outlined by yellow arrowheads) at the segmented layer of the deep vascular plexus. **h** OCTA OS at the same layer for comparison showed normal flow.

further highlighted a potentially blinding complication related to this widely and frequently performed dermatologic procedure in the head and facial region.

The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000543454).

Patient Perspective

I was able to receive attention and monitoring by the retina specialist right away. I am thankful that he immediately communicated with my dermatologist to suspend my planned steroid injections. My vision has returned to close to normal.

Statement of Ethics

At BIDMC, observational studies involving 3 or fewer patients are exempt from Institutional Review Board approval. Written informed consent was obtained from the patient for publication of the details of this medical case and any accompanying images.

Conflict of Interest Statement

The author has no conflicts of interest to declare.

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Author Contributions

Yafeng Li, MD, PhD: conceptualization, data curation, investigation, analysis, methodology, validation, writing – original draft, and writing – review and editing.

Data Availability Statement

All data generated or analyzed during this study are included in this article and its online supplementary material files. Further inquiries can be directed to the corresponding author.

References

1 Ogawa R. Keloid and hypertrophic scars are the result of chronic inflammation in the reticular dermis. IJMS. 2017;18(3):606. https://doi.org/10.3390/ijms18030606
2 Goette DK, Odom RB. Adverse effects of corticosteroids. Cutis. 1979;23(4):477–87.
3 Richards RN. Update on intralesional steroid: focus on dermatoses. J Cutan Med Surg. 2010;14(1):19–23. https://doi.org/10.2310/7750.2009.08082
4 Chavala SH, Williamson JF, Postel EA. Embolic central retinal artery occlusion after subcutaneous auricular steroid injection. Lancet. 2016;387(10034):2235. https://doi.org/10.1016/S0140-6736(15)00980-0
5 Nguyen JD, Duong H. Anatomy, head and neck, posterior auricular artery. StatPearls. Treasure Island (FL): StatPearls Publishing; 2024 [cited 2024 Nov 14]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK546687/

6 Lee JS, Kim JY, Jung C, Woo SJ. Iatrogenic ophthalmic artery occlusion and retinal artery occlusion. Prog Retin Eye Res. 2020;78:100848. https://doi.org/10.1016/j.preteyeres.2020.100848
7 Benzon HT, Chew T-L, McCarthy RJ, Benzon HA, Walega DR. Comparison of the particle sizes of different steroids and the effect of dilution: a review of the relative neurotoxicities of the steroids. Anesthesiology. 2007;106(2):331–8. https://doi.org/10.1097/00000542-200702000-00022
8 Park SSE, Barmettler A. Vision loss secondary to facial and periorbital steroid injection: a systematic review. Ophthalmic Plast Reconstr Surg. 2021;37(6):511–21. https://doi.org/10.1097/IOP.0000000000001910
9 Wahezi S, Mohamed SE, Lederman A, Beck A. Aggregation properties of triamcinolone acetonide injection in human serum: considerations when performing epidural steroid injections. JPR. 2019;12:1033–9. https://doi.org/10.2147/JPR.S181038
10 Cho SC, Jung C, Lee JY, Kim SJ, Park KH, Woo SJ. Retinal artery occlusion after intravascular procedures: case series and literature review. Retina. 2019;39(4):766–78. https://doi.org/10.1097/IAE.0000000000002008
11 Hayreh SS. Prevalent misconceptions about acute retinal vascular occlusive disorders. Prog Retin Eye Res. 2005;24(4):493–519. https://doi.org/10.1016/j.preteyeres.2004.12.001