BMC Ophthalmology

CASE REPORT

Open Access

Vision-threatening complications of injection sclerotherapy: case report, literature review, and FAERS database analysis

Dehai Liu[1†], Xiaona Wang[1†] and Hongliang Dou[1*]

Abstract

Background Injection sclerotherapy using sclerosants such as polidocanol has been widely employed for managing vascular disorders including chronic venous diseases and hemangiomas. Although sclerotherapy is considered a minimally invasive and generally safe procedure, rare but vision-threatening ocular complications have been reported. We present a unique scenario of progressive ophthalmic artery occlusion (OAO) associated with foamed polidocanol injection leading to irreversible blindness in a pediatric patient.

Case presentation A 13-year-old male with a history of facial hemangioma underwent intralesional injection of foamed 1% polidocanol under ultrasound guidance at a dental clinic. He experienced transient monocular blindness in his left eye immediately after injection lasting for approximately 30 min, while best-corrected visual acuity at presentation recovered to 20/20. Despite systemic corticosteroids and anticoagulation, his vision deteriorated to no light perception within 1 month, accompanied by blepharoptosis and cutaneous necrosis. Multimodal imaging including fundus photography, optical coherence tomography, and fluorescein angiography demonstrated progressive retinal vascular occlusion, and Doppler ultrasonography at 1 month identified absent flow in the ophthalmic and central retinal arteries confirming OAO. Furthermore, a global pharmacovigilance analysis revealed that ocular complications represented only 3.9% of reported adverse events for sclerosing agents but were disproportionately severe, with 45.7% classified as death, life-threatening, disabling, or requiring hospitalization; permanent blindness occurred in 6.2% of the total cases.

Conclusions This case underscores the potential for catastrophic ocular complications after polidocanol sclerotherapy. Given the limited therapeutic efficacy once iatrogenic OAO occurs, we emphasize caution when performing sclerosant injections particularly in the risky regions.

Keywords Sclerotherapy, Polidocanol, Complications, Ophthalmic artery occlusion

†Dehai Liu and Xiaona Wang contributed equally to this work.

*Correspondence:
Hongliang Dou
douhl@aliyun.com
[1]Department of Ophthalmology, Beijing Key Laboratory of Restoration of Damaged Ocular Nerve, Peking University Third Hospital, Beijing, China

Background

Over the past several decades, injection sclerotherapy using liquid or foam agents has been demonstrated to be effective and safe for a variety of clinical applications, ranging from a spectrum of chronic vein diseases to hemangiomas and vascular malformations [1, 2]. For example, polidocanol, one of the most commonly used sclerosants, was approved by the US Food and Drug Administration (FDA) for small varicose veins and reticular veins in 2010 [3]. According to the standard medication classifications from RxClass [4], certified sclerosing agents for local injection include polidocanol, sodium tetradecyl sulfate (STS), and ethanolamine oleate. Common adverse events of sclerotherapy are mild and self-limited, including local erythema, injection site pain, superficial cellulitis, and transient allergic responses [3, 5]. Specifically, polidocanol is more likely to be associated with reporting of venous embolic or thrombotic events, STS with allergic reactions, and ethanolamine oleate with cardiac arrhythmias based on an analysis of the World Health Organization pharmacovigilance database VigiBase [6].

Rarely, injection sclerotherapy can lead to severe ocular complications; for example, there existed only a handful of case reports of confirmed ophthalmic artery occlusion (OAO) following local injection of sclerosing agents in the literature to date [7–10] (Table 1). To the best of our knowledge, we described the first pediatric case of progressive OAO following foamed polidocanol sclerotherapy, accompanied by a complementary global pharmacovigilance database analysis to investigate the potential risk, and thus to facilitate early detection and timely management of ocular complications associated with sclerosant injection.

Case presentation

A 13-year-old male with facial hemangioma presented to our emergency department 1 h after receiving an intralesional injection of foamed 1% polidocanol (liquid-to-air ratio 1:3, 23-gauge needle) at a dental clinic. The subcutaneous lesion was located in the left nasolabial region extending toward the upper buccal area. Following a diagnosis of infant hemangioma at age 5, he had received repeated uneventful courses of sclerotherapy throughout

Table 1 Summary of ocular complications reported in the FDA adverse event reporting system database

Characteristics	Total (*N* = 65)	Polidocanol (*N* = 57)	Sodium tetradecyl sulfate (*N* = 7)	Ethanolamine oleate (*N* = 1)
Sex (%)				
Male	14 (25.0)	11 (22.4)	3 (50.0)	-
Female	42 (75.0)	38 (67.6)	3 (50.0)	1
Missing	9	8	1	-
Age group (%)				
<= 18	2 (5.0)	1 (2.9)	1 (20.0)	-
19–39	8 (20.0)	7 (20.6)	1 (20.0)	-
40–59	15 (37.5)	12 (35.3)	2 (40.0)	1
>= 60	15 (37.5)	14 (41.2)	1 (20.0)	-
Missing	25	23	2	-
Region (%)				
Region of the Americas	34 (68.0)	32 (68.1)	2 (66.7)	-
European region	13 (26.0)	13 (27.7)	-	-
Western Pacific region	3 (6.0)	2 (4.2)	1 (33.3)	-
Missing	15	10	4	1
Common reactions (%)				
Visual impairment	31 (47.7%)	27 (47.4%)	4 (57.1%)	-
Visual disturbances	5 (7.7%)	5 (7.7%)	-	-
Abnormal eye sensations	5 (7.7%)	4 (6.2%)	1 (14.3%)	-
Eye swelling	5 (7.7%)	5 (7.7%)	-	-
Anterior segment disorders	5 (7.7%)	5 (7.7%)	-	-
Blindness	4 (6.2%)	2 (3.5%)	2 (28.6%)	-
Blindness transient	4 (6.2%)	4 (6.2%)	-	-
Outcome (%)*				
Serious	21 (45.7)	18 (42.9)	2 (66.7)	1
Non-serious	25 (54.3)	24 (57.1)	1 (33.3)	-
Missing	19	15	4	-

* Serious outcomes reported included life-threatening events, hospitalization, disability, and death

the past three years. During the present session, a slightly larger total dose (4 mL) was injected into the lesion under ultrasound guidance. The patient experienced immediate complete vision loss in the left eye following injection, which lasted for approximately 30 min. During this period, a relative afferent pupillary defect was present, and partial recovery of light perception occurred thereafter. Best-corrected visual acuity at presentation recovered to 20/20. Examination revealed swelling of the left eyelid and maxillofacial region. Both anterior and posterior segment examinations, performed by slit-lamp biomicroscopy and funduscopy respectively, were unremarkable.

Given the uncertain risk of embolism and the early clinical suspicion of retinal vasculitis, the patient was admitted for close observation and prompt intervention. Empirical therapy was initiated with high-dose intravenous methylprednisolone (1,000 mg/day) to suppress potential immune-mediated vascular inflammation. Intravenous Ginkgo biloba extract was administered concurrently to enhance microcirculatory perfusion and support neuronal recovery. Oral rivaroxaban was introduced to prevent secondary thrombosis and maintain vascular patency. The patient tolerated the regimen without adverse effects. Serial fluorescein angiography (FFA) of this patient clearly delineated the progressive course of retinal vascular occlusion following polidocanol injection. Initially, vessel wall staining was evident in the late phase of FFA, affecting both major and minor retinal arteries and veins, suggestive of acute endothelial injury and early vascular compromise (Fig. 1A-B). At 1 week, FFA revealed delayed retinal filling, vascular and optic disc leakage, and focal areas of capillary nonperfusion consistent with early ischemic microangiopathy (Fig. 1C-D). By 1 month, angiography demonstrated markedly delayed choroidal and retinal arterial filling consistent with severe flow reduction, accompanied by pronounced sclerosis of the major vessels, distal vascular hypofluorescence, and extensive capillary nonperfusion—findings indicative of irreversible vascular occlusion and ischemic retinal injury (Fig. 1E-F).

Accordingly, visual acuity fluctuated during treatment and stabilized at 20/25 OS by 1 week; however, two weeks after discontinuation of intravenous therapy, it deteriorated suddenly to no light perception at 1 month, with left-sided blepharoptosis and focal cutaneous necrosis (Fig. 2A). At 1 month, fundus examination revealed diffuse retinal whitening accompanied by optic disc pallor (Fig. 2B), consistent with widespread retinal

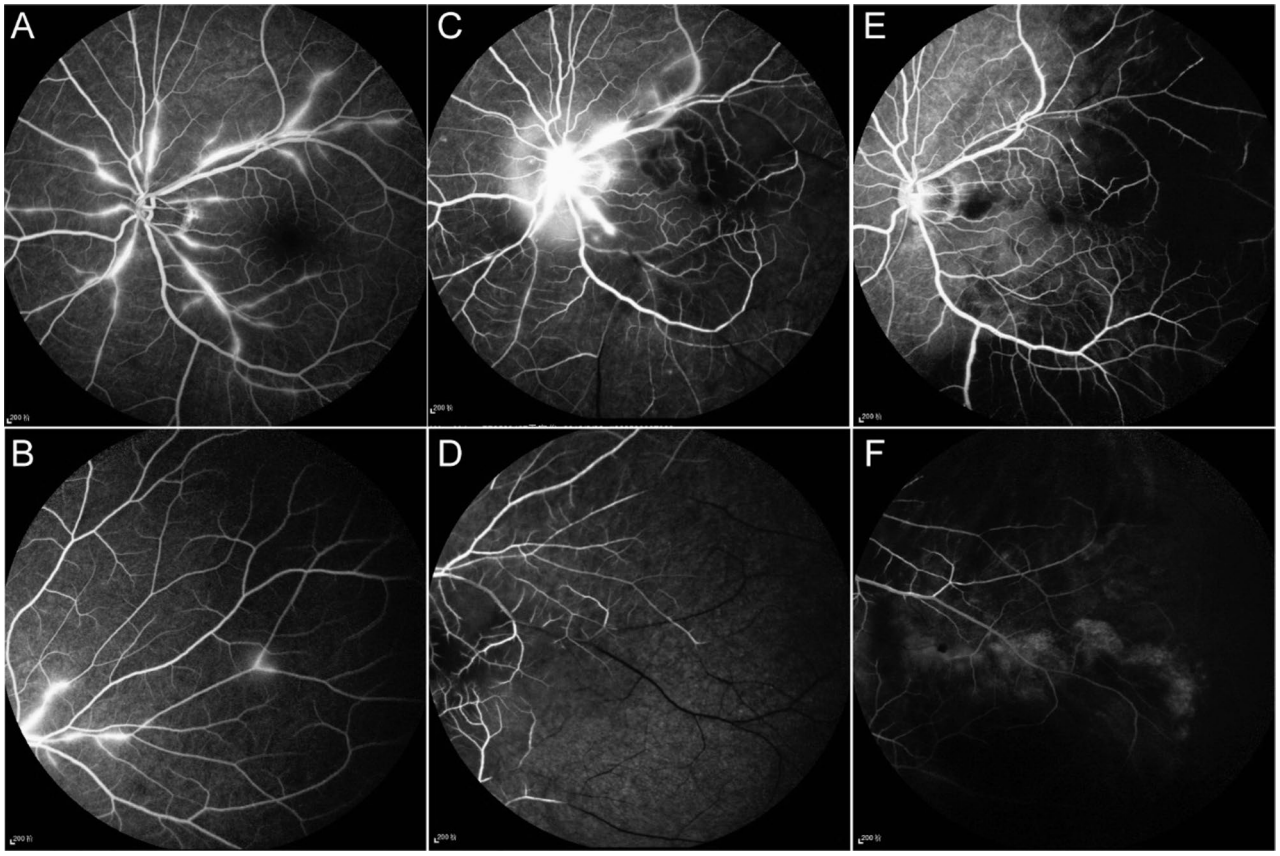

Fig. 1 Fluorescein angiography in late phase taken at 5 min demonstrated vessel wall staining at day 1 (**A**, **B**), vascular and optic disc leakage along with focal capillary nonperfusion at 1 week (**C**, **D**), and distal vascular hypofluorescence and extensive capillary nonperfusion at 1 month (**E**, **F**)

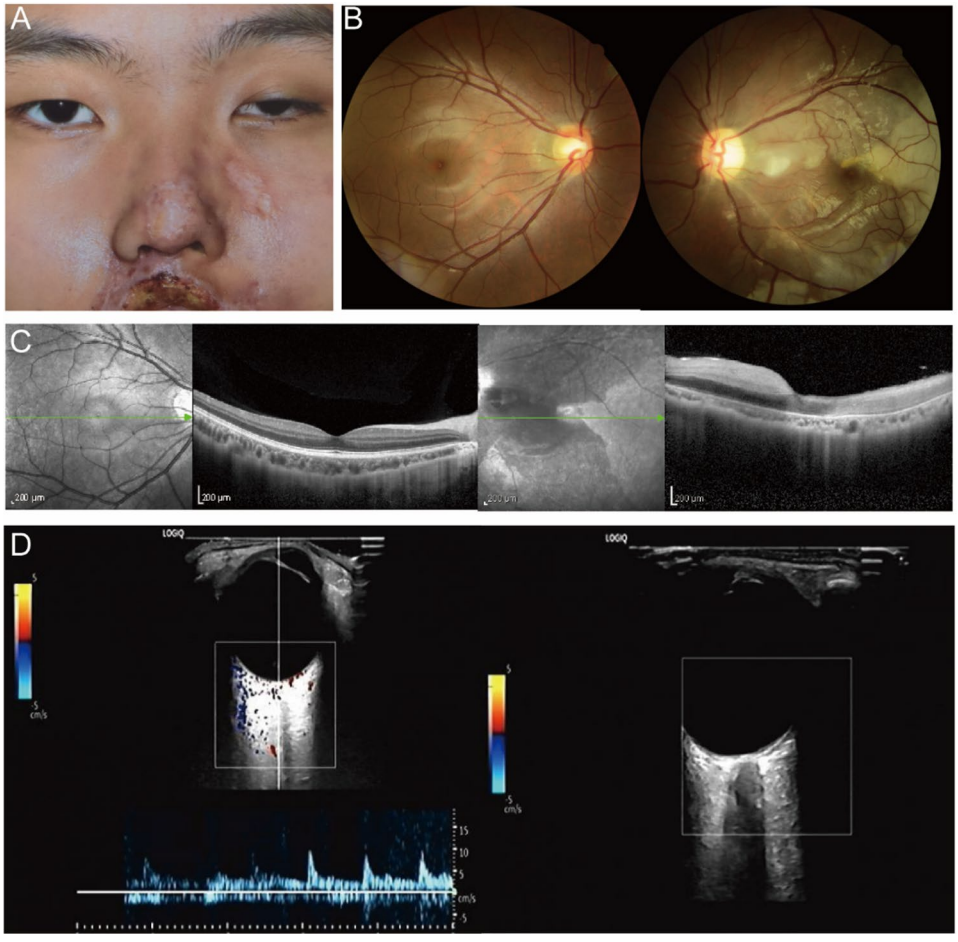

Fig. 2 Examinations at 1 month revealed left-sided blepharoptosis and focal cutaneous necrosis (**A**). Ophthalmic imaging demonstrated panretinal whitening (**B**), hyperreflectivity and thickening of retinal layers (**C**), and absent flow in ophthalmic and central retinal arteries (**D**) in the left eye, and normal fundus (**B**), macular architecture (**C**), and arterial flow (**D**) in the right eye

ischemia. Optical coherence tomography demonstrated pronounced hyperreflectivity across both the inner and outer retinal layers (Fig. 2C), and Doppler ultrasonography at last follow-up identified no flow in left ophthalmic and central retinal arteries (Fig. 2D), thereby confirming the diagnosis of OAO. Brain and carotid imaging were unremarkable. Visual acuity remained no light perception at the 3-month follow-up.

Discussion and conclusions

Injection sclerotherapy is a minimally invasive procedure that induces endothelial injury and subsequent fibrotic occlusion of targeted veins. It is adopted worldwide for chronic venous diseases, with polidocanol among the most frequently used agents [3]. In recent years, the range of off-label indications for sclerotherapy has widened, and sclerosants has been commonly applied as a cost-effective treatment option for hemangiomas, facial veins, and lymphangiomas [5, 11]. Despite a generally favorable safety profile and broad clinical application [12, 13], injection sclerotherapy has been associated with severe ocular complications in sporadic case reports.

Including our case, a total of 5 cases of iatrogenic OAO associated with sclerotherapy were identified in the literature review (Table 1) [7–10]. The etiology was presumed to be polidocanol injection in 2 cases, and STS injection in 3 cases. Foam formulations were used in most cases (80.0%), while only one case reported use of sclerosants in the forms of emulsion. Most reported cases occurred after injections into upper midface regions, particularly the forehead and glabellar areas (80.0%). Patients were primarily composed of young individuals (mean age: 21 years), with left eye involved in all cases. The most common therapeutic options included systemic steroids, oral anticoagulants, hyperbaric oxygen therapy, and anterior chamber paracentesis. Despite these efforts, the final visual acuity was generally poor, and no light perception occurred in at least 80.0% of patients. For example, Matsuo et al. [7] described a case of an 18-year-old male injected with 3% foam polidocanol for glabellar hemangioma who subsequently developed central retinal artery

and posterior ciliary artery occlusion. Visual acuity of this patient rapidly worsened to no light perception on postoperative day 4, and did not recover despite urgent interventions. More recently, Dehghani et al. [9] reported a case of a 16-year-old female undergoing ultrasound-guided intralesional injection of 1% STS using a 23-gauge needle for forehead hemangioma. Immediate complications included dramatic deterioration of visual acuity, and lid swelling of the left eye. Fundus examination at 3-month follow-up revealed ischemic retinal whitening and retinal pigment epithelial disruption, which collectively confirmed the diagnosis of OAO. These findings highlight the aggressive course and limited reversibility of sclerosant-induced OAO.

Similar vision-threatening complications have been reported following cosmetic dermal filler injections into the central and upper midface, which share the similar anatomical risk of retrograde embolization through facial–ophthalmic anastomoses [14]. Although filler procedures are generally considered safe, with most adverse effects limited to bruising, redness, or mild swelling, the growing use of cosmetic injections has led to increasing reports of severe adverse events including monocular blindness. The majority of filler-related OAO have been associated with hyaluronic acid or autologous fat injections, typically performed in the glabellar or nasolabial regions [15]. It has been proposed that filler material can enter the ophthalmic artery via retrograde flow through branches of the superficial temporal artery and its anastomotic branches with the supratrochlear or supraorbital arteries [16]. Patients usually develop sudden visual loss, sometimes accompanied by ipsilateral cerebral infarction or skin necrosis. The visual prognosis is extremely poor: in a pooled analysis of 198 reported cases of vision loss following intravascular filler injection, 58% resulted in no light perception [15]. To date, no therapeutic intervention has been proven to alter the course of filler-related OAO, although preliminary research suggest that intra-arterial thrombolysis may achieve partial recanalization and limited visual recovery [17].

Unlike the previous reports, our case presented a distinctive clinical course characterized by progressive retinal and choroidal vascular occlusion, in contrast to the abrupt onset typically seen in embolic OAO [15]. Transient monocular blindness observed in this patient immediately after injection was likely attributable to gas microembolism, as polidocanol foam produces bubbles measuring 100–1200 μm, sufficient to obstruct the central retinal artery approximately 160 μm in diameter [18, 19]. This microembolic phenomenon resolved spontaneously as gas particles dissipated. However, the subsequent progression to irreversible vascular sclerosis in our patient suggests a dual mechanism of injury: (1) acute retrograde spread and microembolic occlusion followed by (2) sustained downstream arterial propagation and endothelial toxicity of polidocanol, resulting in cumulative ischemic damage [20]. This evolving vascular compromise underscores the possibility that chemical endothelial injury, rather than mechanical embolization alone, may drive the pathophysiology of sclerosant-induced OAO.

To complement our case findings, we further analyzed ocular complications associated with three common sclerosing agents (polidocanol, STS, and ethanolamine oleate) using the FDA Adverse Event Reporting System (FAERS) database, a global pharmacovigilance database of drug safety reports. The search of the FAERS database identified 1,672 adverse event reports related to sclerosing agents for local injection, among which 65 cases (3.9% of total reports) involved ocular complications (Table 2). The majority of cases were associated with polidocanol ($n = 57$), with the remaining cases attributed to STS ($n = 7$) and ethanolamine oleate ($n = 1$). After excluding 27 cases with unspecified indications, the most

Table 2 Clinical profiles of patients diagnosed with ophthalmic artery occlusion following injection sclerotherapy in the literature

Author (year)	Age, year	Gender	Eye	Sclerosing agents	Indication	Treatment	Follow-up	Visual acuity at last visit
Our case (2025)	13	Male	OS	polidocanol foam, 23-gauge needle	nasolabial hemangioma	systemic steroids, Ginkgo biloba extract, and oral anticoagulants	3 months	NLP
Arunakirinathan (2019) [10]	26	Female	OS	sodium tetradecyl sulfate foam	forehead vein	intravenous acetazolamide, carbon dioxide rebreathing, ocular massage, and anterior chamber paracentesis	3 months	NLP
Dehghani (2018) [9]	16	Female	OS	sodium tetradecyl sulfate emulsion, 23-gauge needle	forehead hemangioma	oral acetazolamide, anterior chamber paracentesis, and systemic steroids	3 months	NLP
Esmaili (2017) [8]	33	Female	OS	sodium tetradecyl sulfate foam	forehead vein	hyperbaric oxygen	NA	HM
Matsuo (2009) [7]	18	Male	OS	polidocanol foam, 23-gauge needle	glabellar hemangioma	systemic steroids, oral anticoagulants, and hyperbaric oxygen	2.5 years	NLP

* NLP: no light perception; HM: hand motion; NA: none-available

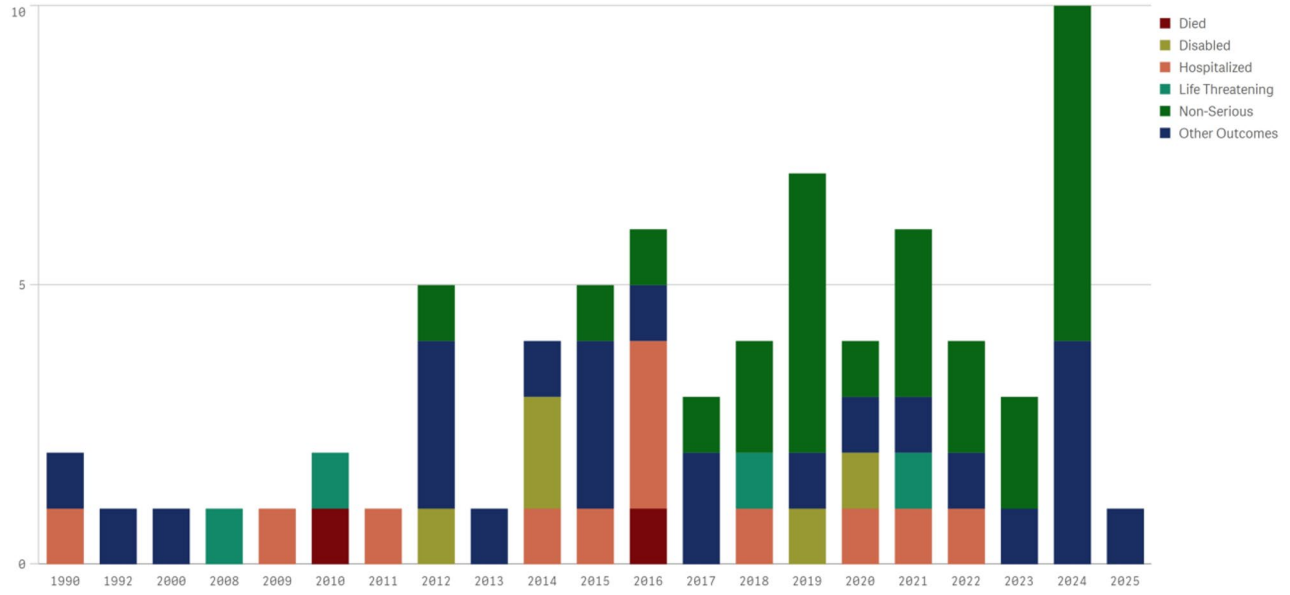

Fig. 3 Patient outcomes associated with injection sclerotherapy in the FDA adverse event reporting system database

frequent diagnosis was chronic venous disease ($n = 34$) involving varicose veins and spider veins. The remaining cases involved vascular malformation ($n = 2$) and lymphangioma ($n = 2$). The majority of the patients were female (75.0%), aged ≥ 40 years (75.0%), and from the Americas region (68.0%). Ocular complications represented a relatively uncommon subset of adverse events, yet notably severe with 45.7% of the cases meeting criteria for death, life-threatening, disabling, or hospitalization outcomes (Fig. 3). Specifically, there existed 2 cases of deaths, 3 of life-threatening events, 5 of disabilities, and 11 of hospitalizations. Among adverse reactions reported by FAERS database, the most common ocular manifestations were visual impairment (47.7%), followed by visual disturbances, abnormal ocular sensations, eye swelling, and anterior segment disorders, with each representing 7.7% of reported cases respectively. Notably, permanent blindness was observed in 6.2% of all cases (Table 2). These findings indicate that ocular complications of sclerotherapy, though rare, carry an unusually high risk of irreversible visual loss. It should be noted, however, that the FAERS database is a voluntary reporting system prone to under-reporting and reporting bias, with minor events less likely and severe complications more likely to be reported, potentially inflating the apparent severity rate. Therefore, these data should be interpreted with caution.

The underlying mechanisms of vision loss are likely multifactorial. Embolic occlusion by sclerosant microbubbles or particulate matter has been proposed as a primary cause [21]. Retrograde flow through arteriovenous anastomoses allows the sclerosant entry into the ophthalmic circulation. Beyond mechanical obstruction, sclerosants such as polidocanol and STS exert direct endothelial toxicity, causing apoptosis, vasospasm, and inflammatory changes in the vessel wall [5]. Interestingly, emerging evidence suggests that biochemical mediators may also contribute. For instance, elevated plasma levels of endothelin-1, a potent vasoconstrictor, have been observed following foam sclerotherapy, which may correlate with transient visual disturbances reported in some patients [22]. Indeed, in the acute setting, distinguishing between embolic and chemically toxic mechanisms of occlusion is clinically challenging but may influence therapeutic decision-making. For example, embolic occlusion may prompt attempts at reperfusion, whereas chemically induced vasospasm and endothelial injury are less likely to respond to such interventions. Some authors have therefore proposed systemic corticosteroids in cases of suspected vasculitis associated with circulating toxic agents, but evidence supporting these approaches is lacking. This uncertainty underscores the difficulty of formulating effective reactive treatments and reinforces the importance of prevention.

Therapeutic options for OAO remain limited and largely ineffective once irreversible ischemia is established. For embolic OAO, several emergency interventions have been attempted [16]: (1) dilating the retinal arteries by vasodilators, and lowering intraocular pressure by medications, anterior chamber paracentesis or ocular massage to increase perfusion pressure, (2) endovascular thrombolysis (e.g., selective intra-arterial fibrinolysis) for direct arterial recanalization and systemic anticoagulation (e.g., oral anticoagulants) to prevent thrombus propagation, and (3) hyperbaric oxygen therapy and intravenous corticosteroids for anti-inflammatory and neuroprotective effects. However, the efficiency

of these treatment modalities to improve prognosis of OAO still remains limited, primarily hampered by the narrow ischemic survival window of retinal ganglion cells which were estimated at 15 min [23]. Additionally, there is no evidence that they are effective in chemically induced arterial obstruction. Prevention through meticulous technique remains the only reliable strategy. This may explain the consistent poor visual outcomes documented in both our case and prior reports.

Given the therapeutic limitations, we underscore the importance of prevention strategies especially for patients at increased risk. Clinicians must recognize that local facial injections carry unique risks due to anastomoses between branches of the external carotid and ophthalmic arteries. Retrograde flow of liquid agents or foam bubbles can readily compromise retinal circulation during etiologically associated medical procedures such as cosmetic facial filler injection and sclerotherapy [15]. While ultrasound guidance cannot fully prevent retrograde propagation of sclerosant via collateral networks, it remains one of the most important risk-reduction measures and should be considered mandatory when treating lesions in high-risk anatomical zones [21]. We recommend preventive measures including lowest effective concentration and volume, controlled injection speed and pressure, as well as multidisciplinary team support particularly in pediatric or otherwise vulnerable cases [13].

In conclusion, ocular complications of injection sclerotherapy were rare with a significant proportion progressing to severe adverse events that lead to poor visual prognosis. Our case illustrates a unique scenario of progressive OAO following polidocanol injection likely mediated by a combination of microembolic and chemical endothelial injury. Together with pharmacovigilance data, these findings underscore the urgent need for precise anatomical understanding, careful procedural planning, and heightened vigilance during sclerosant injections particularly in patients at increased risk.

Abbreviations

OAO	Ophthalmic artery occlusion
FDA	Food and drug administration
STS	Sodium tetradecyl sulfate
FFA	Fluorescein angiography
FAERS	FDA Adverse event reporting system

Acknowledgements

We would like to thank the anonymous editors and reviewers for their valuable suggestions for improving the quality of this article.

Author contributions

D.L. and X.W. contributed equally to this work. X.W. and H.D. performed the ophthalmic examinations for the patient. X.W. and D.L. collected the clinical information of the patient, analyzed and interpreted the clinical data. D.L. and X.W. were major contributors to the writing of the manuscript. H.D. reviewed and edited the manuscript. All the authors read and approved the final manuscript.

Funding

Not applicable.

Data availability

All the data and materials supporting our findings are contained within this manuscript.

Declarations

Ethics approval and consent to participate

This study was performed in accordance with the tenets of the Declaration of Helsinki. Consent to participate was not applicable due to the retrospective design of this study.

Consent for publication

Written informed consent was obtained from the parents of the patient for the publication of this report and the accompanying images.

Competing interests

The authors declare no competing interests.

Received: 8 September 2025 / Accepted: 14 November 2025
Published online: 27 November 2025

References

1. de Ávila Oliveira R, Riera R, Vasconcelos V, Baptista-Silva JC. Injection sclerotherapy for varicose veins. Cochrane Database Syst Rev Dec. 2021;10(12):Cd001732. https://doi.org/10.1002/14651858.CD001732.pub3.
2. Gao Z, Zhang Y, Li W, Shi C. Effectiveness and safety of polidocanol for the treatment of hemangiomas and vascular malformations: a meta-analysis. Dermatol Ther Jan. 2018;31(1). https://doi.org/10.1111/dth.12568.
3. Hamdan A. Management of varicose veins and venous insufficiency. Jama Dec. 2012;26(24):2612–21. https://doi.org/10.1001/jama.2012.111352.
4. RxClass browser version: 3.1.168. Accessed August. 6th, 2025. https://mor.nlm.nih.gov/RxClass/
5. Nguyen QD, Stender C, Bur D, Silapunt S, Polidocanol. A review of Off-Label dermatologic uses. Dermatol Surg Sep. 2022;1(9):961–6. https://doi.org/10.1097/dss.0000000000003520.
6. Roselli A, Khouri C, Roustit M, Blaise S, Cracowski JL. Safety profile of sclerosing agents: an analysis from the world health organization pharmacovigilance database vigibase. Dermatol Surg Dec. 2019;45(12):1517–28. https://doi.org/10.1097/dss.0000000000001876.
7. Matsuo T, Fujiwara H, Gobara H, Mimura H, Kanazawa S. Central retinal and posterior ciliary artery occlusion after intralesional injection of sclerosant to glabellar subcutaneous hemangioma. Cardiovasc Intervent Radiol Mar. 2009;32(2):341–6. https://doi.org/10.1007/s00270-008-9382-9.
8. Esmaili DD. Iatrogenic occlusion of the ophthalmic artery after sodium tetradecyl sulfate, injection in the forehead. Retin Cases Brief Rep Winter. 2017;11(Suppl 1):S28–30. https://doi.org/10.1097/icb.0000000000000393.
9. Dehghani A, Rezaei L, Ghanbari H, Nasrollahi K, Tavakoli M. Ophthalmic artery occlusion following facial sclerosing therapy. J Ophthalmic Vis Res Jul-Sep. 2018;13(3):351–4. https://doi.org/10.4103/jovr.jovr_29_16.
10. Arunakirinathan M, Walker RJE, Hassan N, Ameen S, Younis S. Blind-sided by cosmetic vein, sclerotherapy: a case of ophthalmic arterial occlusion. Retin Cases Brief Rep Spring. 2019;13(2):185–8. https://doi.org/10.1097/icb.0000000000000559.
11. Yang C, Li M, Li X, Zhu J, Shu C. Foam sclerotherapy in the treatment of hemangiomas and venous malformations. Dermatol Surg Sep. 2023;1(9):855–61. https://doi.org/10.1097/dss.0000000000003857.
12. Brittenden J, Cotton SC, Elders A, et al. Clinical effectiveness and cost-effectiveness of foam sclerotherapy, endovenous laser ablation and surgery for varicose veins: results from the comparison of LAser, surgery and foam sclerotherapy (CLASS) randomised controlled trial. Health Technol Assess Apr. 2015;19(27):1–342. https://doi.org/10.3310/hta19270.
13. De Maria L, De Sanctis P, Tollefson M, et al. Sclerotherapy for low-flow vascular malformations of the orbital and periocular regions: systematic review and meta-analysis. Surv Ophthalmol Jan-Feb. 2020;65(1):41–7. https://doi.org/10.1016/j.survophthal.2019.08.003.

14. Wu S, Pan L, Wu H, et al. Anatomic study of ophthalmic artery embolism following cosmetic injection. J Craniofac Surg Sep. 2017;28(6):1578–81. https://doi.org/10.1097/scs.0000000000003674.
15. Foster J, Aakalu VK, Freitag SK, et al. Vision-Threatening complications of soft tissue fillers: a report by the American academy of ophthalmology. Ophthalmol Aug. 2025;132(8):935–44. https://doi.org/10.1016/j.ophtha.2025.01.020.
16. Lee JS, Kim JY, Jung C, Woo SJ. Iatrogenic ophthalmic artery occlusion and retinal artery occlusion. Prog Retin Eye Res Mar. 2020;10:100848. https://doi.org/10.1016/j.preteyeres.2020.100848.
17. Wang J, Shen H, Liu T, Li Q, Lyu Z, Yu Y. An efficacy and safety study of Intra-arterial recanalization of occluded ophthalmic arteries in patients with monocular blindness caused by injection of hyaluronic acid in facial tissues. Aesthetic Plast Surg Aug. 2021;45(4):1573–8. https://doi.org/10.1007/s00266-021-02224-4.
18. Nastasa V, Samaras K, Ampatzidis C, et al. Properties of polidocanol foam in view of its use in sclerotherapy. Int J Pharm Jan. 2015;30(2):588–96. https://doi.org/10.1016/j.ijpharm.2014.11.056.
19. Wong K, Connor DE, Andrade OC, Parsi K. Foam bubble size is significantly influenced by sclerosant concentration for polidocanol but not sodium tetradecyl sulphate. Phlebology Aug. 2021;36(7):576–87. https://doi.org/10.1177/0268355521995012.
20. Raufi NN, Wisely CE, Leyngold IM, Vajzovic L. Ophthalmic and central retinal artery occlusion following triamcinolone injection of the lacrimal gland. JAMA Ophthalmol Dec. 2019;1(12):1460–1. https://doi.org/10.1001/jamaophthalmol.2019.3998.
21. Gillet J. Complications and side effects: European guidelines for sclerotherapy in chronic venous disorders. Phlebology May. 2014;29(1 suppl):34–8. https://doi.org/10.1177/0268355514526532.
22. Frullini A, Barsotti MC, Santoni T, Duranti E, Burchielli S, Di Stefano R. Significant endothelin release in patients treated with foam sclerotherapy. Dermatol Surg May. 2012;38(5):741–7. https://doi.org/10.1111/j.1524-4725.2012.02390.x.
23. Tobalem S, Schutz JS, Chronopoulos A. Central retinal artery occlusion - rethinking retinal survival time. BMC Ophthalmol Apr. 2018;18(1):101. https://doi.org/10.1186/s12886-018-0768-4.

Publisher's note