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Case Report

A Case of Traumatic Submacular Hemorrhage Treated with tPA and Pneumatic Displacement

Lorenzo Motta[a] Matteo Ripa[a] Korina Theodoraki[b] Timothy L. Jackson[b, c] Dominic McHugh[b]

[a]Department of Ophthalmology, William Harvey Hospital, East Kent Hospitals University NHS Foundation Trust, Ashford, UK; [b]Department of Ophthalmology, King's College Hospital NHS Foundation Trust, London, UK; [c]King's Ophthalmology Research Unit, School of Life Sciences and Medicine, King's College London, London, UK

Keywords

Orbital floor fracture · Pneumatic displacement · Submacular hemorrhage · Tissue plasminogen activator · Trauma

Abstract

This is a case of a 31-year-old female who presented to the emergency department at a London teaching hospital with a 24-h history of visual loss following an assault. The ophthalmological routine examination showed a submacular hemorrhage (SMH), and a computerized tomography scan demonstrated a displaced orbital floor fracture with inferior rectus entrapment and a medial wall fracture. To induce displacement of the SMH, intravitreal injection of 0.25 µg tissue plasminogen activator (tPA) was combined with 0.3 mL of intravitreal 100% perfluoropropane (C_3F_8) gas. At the 1-day follow-up, there was an inferotemporal displacement of the blood clot, and visual acuity improved from hand motions to 6/5 within 3 months. No complications occurred over 2 years of follow-up, with a final visual acuity of 6/5. This case shows us that intravitreal tPA and gas appear safe and effective as a treatment for traumatic SMHs. Furthermore, our results demonstrate that prompt treatment leads to favorable anatomical and functional outcomes.

Lorenzo Motta and Matteo Ripa contributed equally to this work and should be considered equally first authors.

Correspondence to:
Matteo Ripa, matteof12@gmail.com

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Introduction

Ocular trauma can cause severe disability and visual loss. In the USA, it has been estimated that 6.98 per 1,000 population present to the emergency department with an eye injury [1]. The socio-economic impact is substantial, especially since most of those injuries involve those of working age. Blunt trauma is associated with retinal and subretinal hemorrhages, commotio retina, choroidal rupture, and macular holes [2, 3]. These injuries can cause severe visual dysfunction, submacular hemorrhage (SMH), and irreversible damage to the retinal pigment epithelium and the outer neuroretina [4].

Case Report

A 31-year-old female presented to the emergency department at a London teaching hospital with a 24-h history of visual loss following an assault, wherein she sustained blunt trauma to her left eye. Presenting best-correct visual acuity was hand movements. Gonioscopy showed no signs of angle recession, and the intraocular pressure was normal. There was no traumatic mydriasis, iris dialysis, or phacodonesis. There was mild chemosis and corneal abrasion. Left enophthalmos and malar numbness were noted. Dilated fundoscopy revealed a left SMH as shown in Figure 1. Swept source optical coherence tomography showed diffuse submacular hyporeflective material with a neurosensory retinal detachment with no Bruch's membrane breaks (shown in Fig. 2).

A computerized tomography scan demonstrated a displaced orbital floor fracture with inferior rectus entrapment and a medial wall fracture. She was referred to the maxillofacial team and underwent surgical repair.

To induce displacement of the SMH, intravitreal injection of 0.25 μg tissue plasminogen activator (tPA) was combined with 0.3 mL of intravitreal 100% perfluoropropane (C_3F_8) gas. Afterward, face-down positioning for 45 min every hour per week during waking hours was required to support blood displacement. One day after the procedure, the best-correct visual acuity improved to 6/18, a 10% intravitreal gas bubble was present, and the clot was displaced temporally and reduced in size.

Six weeks post-injections, the vision improved further to 6/12 and 6/5 at 3 months. There was a hyperreflective area in the OCT representing the previous location of the clot in the inferotemporal posterior pole. Additional treatment was not required, including anti-vascular endothelial growth factor (VEGF) therapy, and there were no further visual symptoms until the final visit 2 years after the procedure.

All medical and surgical procedures were carried out following the principles of the Declaration of Helsinki. The patient provided written informed consent for using her clinical records and any accompanying image for research purposes. The CARE Checklist has been completed by the authors for this case report and is attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000534199).

Discussion

SMH may be classified according to its size: small, less than four disc diameters, medium, greater than four disc diameters but does not extend beyond the temporal vascular arcade, and a massive SMH that extends beyond the temporal vascular arcade [5]. Speed of treatment may be important. Glatt and Machemer [6] injected fresh autologous blood into the subretinal

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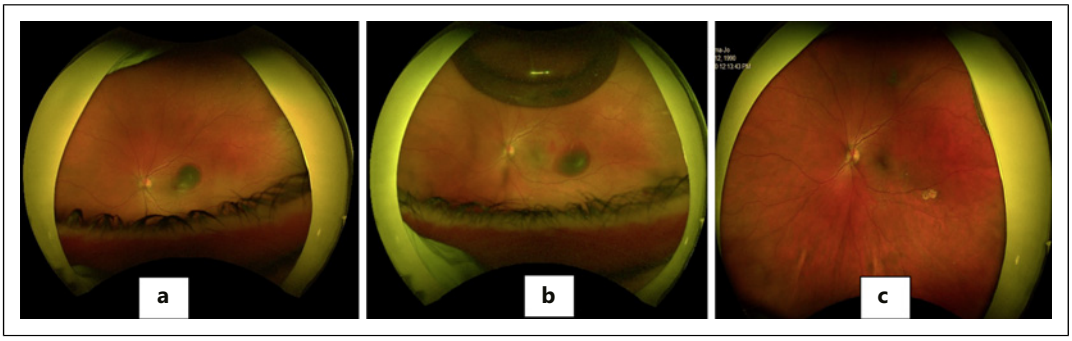

Fig. 1. Color fundus photo of the left eye showing increased macular thickness with the presence of submacular hemorrhage (SMH) at presentation (**a**), minimal reduction of the SMH at 1-day follow-up with blood clot displaced temporally to the fovea (**b**), and resolved SMH at 3-month follow-up (**c**).

space in a rabbit model and detected photoreceptor damage just 1 h after injury, with further photoreceptor damage over the following 7 days. Photoreceptor changes included edema, associated with pyknosis and karyolysis of the outer nuclear layer.

Multiple mechanisms may contribute to macular damage. As the blood clot dissolves, the red blood cells are phagocytized by macrophages. Hemosiderin is converted to retinotoxic ferritin, leading to oxidative stress and permanent damage to the outer nuclear layer and retinal pigment epithelium. In addition, the clot stimulates chemoattractants and fibroblasts, leading to scar formation and retraction, which may induce photoreceptor avulsion [7].

Current treatment modalities include intravitreal or subretinal injection of tPA, pneumatic displacement, and a combination thereof. Intravitreal anti-VEGF injections are also administered with pneumatic displacement, but usually when there is an underlying VEGF-drive cause such as neovascular age-related macular degeneration [8]. tPA's thrombolytic effect catalyzes the conversion of plasminogen to plasmin, degrading fibrin and thereby reducing the fibrin-mediated blood clot adherence, with subsequent further mitigation of retinal damage.

The use of intravitreal tPA and expansile gas has been widely investigated, and several studies have concluded that 25–50 μg per 0.1 mL is the safe and effective dose for intravitreal use as higher doses have been associated with ocular complications and further retinal damage as reported by Chen et al. [9].

TPA can also be injected into the subretinal space (0.1 mL via a 41-gauge needle) during a pars plana vitrectomy. Although more technically demanding, subretinal injection of tPA followed by gas tamponade may be more effective in terms of the visual outcome when compared to intravitreal tPA injection. However, definitive studies are lacking and surgical releases of posterior vitreous detachment in young patients can be difficult and result in retinal tears [10].

The use of anti-VEGF for traumatic SMH was first reported by Abdul-Salim et al. [11], who, in 2013, treated a 23-year-old man with SMH secondary to blunt ocular trauma with a single dose of 0.5 mg of intravitreal ranibizumab. Although the exact mechanism of action is unknown, anti-VEGF might reduce vascular permeability, boosting blood resorption.

The timing of treatment is crucial in resolving SMH, and the literature supports early intervention as poor outcomes have been reported if the treatment is delayed more than a week [12–14]. Indeed, eyes with a duration of SMH of less than 7 days have a significantly higher probability of achieving anatomical and functional favorable outcomes than those with older SMH.

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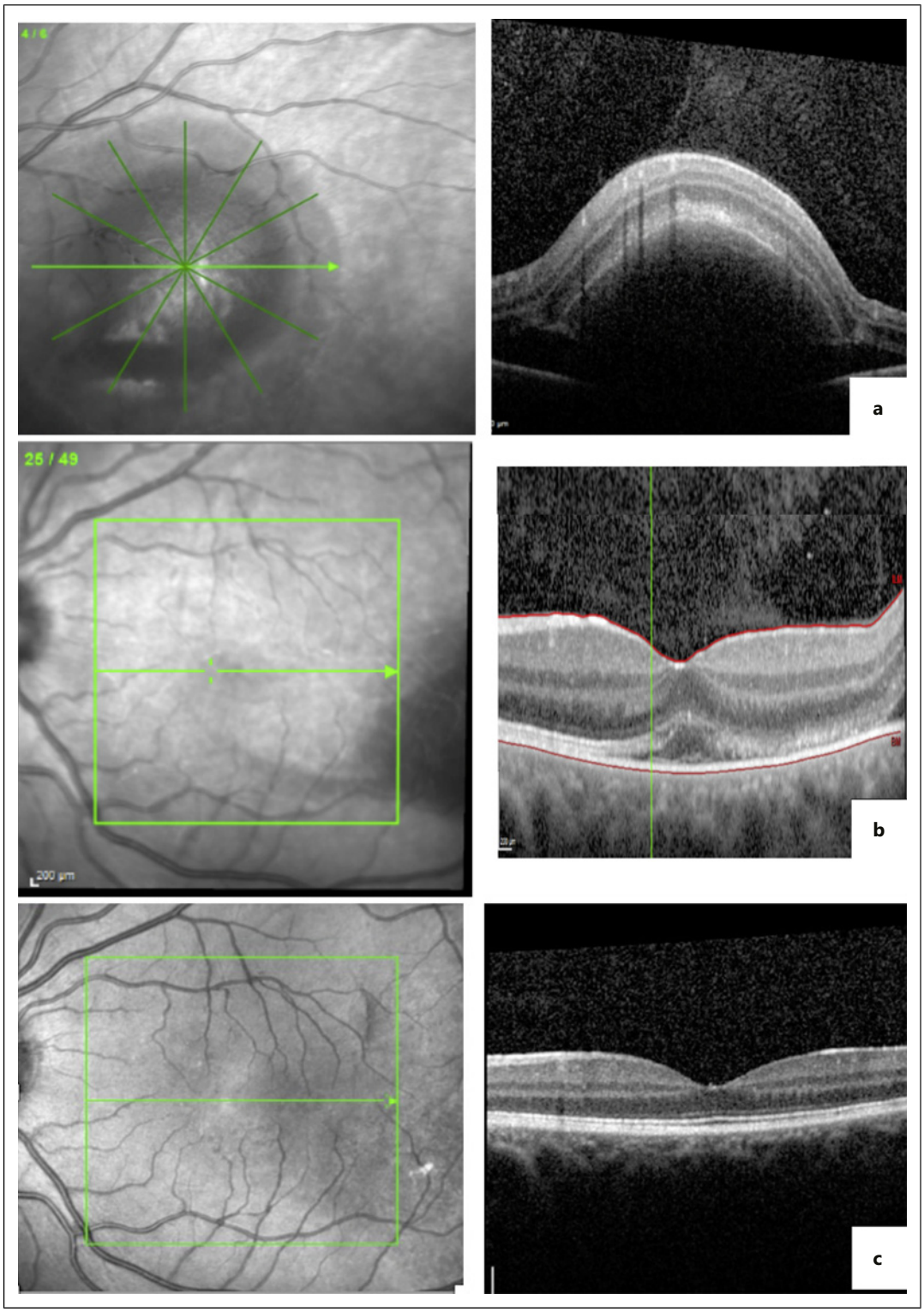

Fig. 2. Optical coherence tomography of the left eye shows diffuse submacular hyporeflective material with a neurosensory retinal detachment suggestive of submacular hemorrhage (SMH) at the presentation (**a**), minimal reduction of the SMH at 1-day follow-up with blood clot displaced temporally to the fovea (**b**), and resolved SMH at 3-month follow-up (**c**).

In our case, we administered intravitreal tPA and C_3F_8 within 48 h of the injury. There was an excellent anatomical and functional outcome with full visual recovery. Therefore, our results support the importance of timing in managing SMH.

In conclusion, traumatic SMH should be managed as an ophthalmic emergency, and patients should be offered intervention within days of the presentation. If a vitreoretinal service is not available locally, a referral should be made to the nearest unit. Delay in treatment can compromise the visual prognosis [7].

Statement of Ethics

The authors endorse that the patient has given their written informed consent to publish their case (including publication of images). Ethical approval for this study is not required in accordance with local or national guidelines.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

Conceptualization: L.M., M.R., and T.J.; methodology, software, data curation, visualization, and project administration: M.R.; validation: T.J. and L.M.; formal analysis, investigation, writing – original draft preparation: M.R. and K.T.; resources: L.M., K.T., T.J., D.M.; writing – review and editing: M.R., T.J, and D.M.; supervision, M.R., T.J., and D.M.; funding acquisition, none. All authors have read and agreed to the published version of the manuscript.

Data Availability Statement

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

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