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CASE REPORT

# Spontaneous expulsive suprachoroidal haemorrhage in an asymptomatic elderly patient: a case report

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**Abstract**
*Purpose* To describe a rare case of spontaneous expulsive suprachoroidal haemorrhage (SESCH) in an asymptomatic elderly patient.
*Method* This is a case report of a 76-year-old Chinese female, presented as an emergency with spontaneous left eye bleeding. She had underlying uncontrolled hypertension, no other systemic illness and not on anticoagulant. She has a history of right eye cataract operation, right eye angle-closure glaucoma and left eye absolute glaucoma complicated with painless left blind eye. Ocular examination over left eye showed no light perception and demonstrated presence of fresh bleed, expulsion of lens and prolapsed uveal contents, while right eye examination was unremarkable. Patient subsequently underwent evisceration and was uneventful.
*Results* Routine blood investigations including coagulation profile came back as normal. Surgical findings include perforated cornea more than three-fourths with prolapsed uveal contents and fragile conjunctiva. No other significant macroscopic conditions were noted. Histology and culture came back with growth of Pseudomonas aeuroginosa with no evidence of malignancy.
*Discussion* SESCH is a rare but serious sight-threatening ocular condition associated with multiple risk factors including arteriosclerosis, vascular disease, glaucoma, diabetes, intraocular malignancy and diseased eye wall. The predisposing factors involved in this case include advanced age, glaucoma with persistent high intraocular pressure, uncontrolled hypertension and presence of infection.

**Keywords** Spontaneous expulsive suprachoroidal haemorrhage · Glaucoma · Cornea damage · Globe rupture · Hypertension · Asymptomatic · Elderly patient

## Introduction

Spontaneous expulsive suprachoroidal haemorrhage (SESCH) is a rare but serious sight-threatening ocular condition. Expulsion of the intraocular contents occurs through the anterior eye wall, leading to devastating outcome. Only a handful reported this condition [1–3]. There are multiple predisposing factors of SESCH, including age more than 60 years, arteriosclerosis, hypertension, diabetes, vascular disease, glaucoma, aphakia or pseudophakia and intraocular malignancies [3]. This article presents a case of a patient with multiple risk factors and presented with SESCH and

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**Fig. 1** Patient's condition upon arrival at the emergency room

discusses the assessment, diagnosis and management for this patient (Figs. 1, 2).

## Case report

A 76-year-old Chinese female presented as an emergency with left eye spontaneous bleeding. She has underlying hypertension, taking oral Amlodipine at a 5 mg daily dose. Otherwise, she has no other systemic illness and not on anticoagulant. She had her right eye cataract operation done 10 years ago. Two days prior to the presentation, she experienced tolerable headache with mild left eye pain. On the day of presentation, she described bleeding from her left eye while resting. Patient denied history of trauma of any nature as well as other systemic symptoms. Nearly 5 years ago, she was diagnosed with right eye chronic angle-closure glaucoma and left eye absolute glaucoma complicated with painless left blind eye and has been under our regular follow-up. She was well informed of her diagnosis and was on three types of anti glaucoma medications for her right eye, namely topical Timolol twice daily, topical Dorzolamide eight hourly, topical Latanoprost once daily and only on lubricating eye drops for her left eye. Throughout her follow-up in our hospital, it had been noted that her left eye intraocular pressure (IOP) had been chronically high with presence of cornea scar. During her last follow-up in our hospital, it was documented that her left eye IOP was 46 mmHg.

At presentation, her blood pressure was high (182/98 mmHg) and required immediate attention, which she was given oral Nifedipine 5 mg single dose.

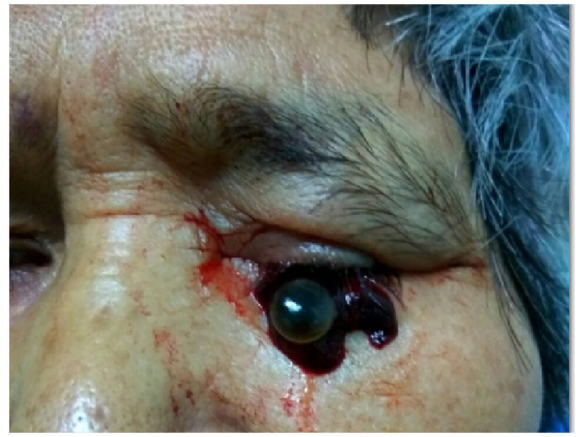

**Fig. 2** Acutely prolapsed lens

Ocular examination revealed no light perception over the affected eye and 6/12 in the fellow eye. There was expulsion of lens over left eye and prolapsed uveal contents. Furthermore, fresh bleeding was also noted and pressure patch was applied which was fully soaked. Right eye examination was unremarkable with a normal IOP and a patent peripheral iridotomy. Fundoscopy over the right eye revealed pale optic disc with a disc cup ratio of 0.4 and flat macula.

Blood investigations including coagulation profile came back as normal, and evisceration was planned the next morning. The intervention was performed with general anaesthesia and was uneventful. There were no signs of other form of macroscopic conditions of the eyeball. Surgical findings included a perforated cornea more than three-fourths and hanging only by one fourth on the circumference with prolapsed uveal tissues and fragile conjunctiva. Besides, blood clot from the suprachoroidal haemorrhage was noted. Acrylic ball was not implanted after discussion with patient and family members. Histology and culture noted the presence of Pseudomonas aeuroginosa, and patient was treated with oral Ciprofloxacin for total duration of 10 days. No evidence of malignancy was noted. Patient was subsequently discharged.

## Discussion

This case of SESCH stands out as it might be caused by a combination of multiple risk factors, namely advanced age, underlying glaucoma with persistent high IOP, uncontrolled hypertension and presence of

infection. The exact mechanism causing SESCH has been a favourable topic for discussion at various scientific meetings. In this case, there are three possible mechanisms of SESCH in this case as dictated by the patient's risk factors, namely inflammatory necrosis of the choroidal vessel wall [4], sudden compression and decompression events [5] and role of infection.

A lion's share of the cases reported involved patients with underlying glaucoma and cornea damage to a certain extent. Likewise, our patient has left eye absolute glaucoma in addition with cornea scaring and underlying hypertension; together, they increase her likelihood of local ischaemia of the fragile vessels. Local ischaemia can cause necrosis of the posterior ciliary arteries and with cornea damage, this consequence in the development of corneal perforation. Any corneal or sclera perforation results in sudden ocular decompression with anterior shifting of the retina and choroid subsequently might cause rupture of the posterior ciliary arteries and ensued by suprachoroidal haemorrhage [6]. Histopathology study reported by Wolter and Garfinkel illustrated that the long posterior ciliary arteries appear especially vulnerable to rupture because their connections between the scleral exit and the outer choroid are short [7].

Furthermore, Perry et al. [8] reported a case of a patient with absolute glaucoma in which the cause, from the histology report, would be an inflammatory necrosis that possibly affected the choroid and vessels. In addition, Sudhir et al. [9] reported a case of SESCH, which was postulated that an elevated IOP with weakening of cornea was responsible for the rupture. In glaucomatous eyes, higher IOP can decrease the nutrient supply to the vessels, accentuating necrosis of the vessel walls. Thus, this sheds some light regarding optimizing both medical and surgical treatment in patients with absolute glaucoma. In our case, patient is only on lubricating drops over her left eye despite chronically high IOP. This raised an interesting point regarding the concept of individualized target IOP and brought us to question whether this patient's outcome would be any different if we have targeted a more aggressive reduction in the IOP. However, we also need to take into account underlying conditions that can interfere with the compliance including economic and social issues. In our hospital setting, economic and social problems may also limit patients' access to medical treatment for glaucoma.

Pollack et al. [10] have suggested that a sudden increase in systemic blood pressure results in an increase in episcleral vascular resistance and IOP, which is further transmitted to the vulnerable choroidal vessels. This can lead to sudden compression and decompression of the fragile vessels, causing rupture and followed by suprachoroidal haemorrhage. Similarly, our patient has uncontrolled hypertension and initially presented with a high blood pressure that requires immediate attention. Hence, there is a high possibility that SESCH in this case was initiated by sudden surge in the systemic pressure.

Infection also plays a pivotal role in SESCH. In 2011, Park et al. [11] reported a case of a 94-year-old lady with massive suprachoroidal haemorrhage and spontaneous eyeball rupture, in which the cause was thought to be thin corneal limbus due to infection, complicated with acute angle-closure glaucoma and caused massive rupture. Comparably, presence of Pseudomonas aeuroginosa in this case suggested infection to some extent which might have contributed to cornea ulcer despite pre existing corneal scarring. Ophir et al. [1] also suggested that difference in elasticity of the cornea at the limbus makes it more vulnerable to perforation.

We report a clinical case of SESCH as we acknowledge that the incidence is extremely rare and is one of the very few cases reported where patient is not on anticoagulants. We believe that the cause of SESCH in our case is due to a combination of glaucoma with persistent high IOP, in addition to her uncontrolled hypertension and presence of infection which lead to subsequent development of complications as described above. Thorough understanding of the risk factors, pathophysiology and possible outcome allow optimization and improvement in our management to avoid the outcome of SESCH which affects physically and emotionally.

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**Compliance with ethical standards**

**Conflict of Interest** The authors declare that they have no conflict of interest.

**Informed Consent** Informed consent was obtained from the patient for publication of this case report and accompanying images.

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