



CASE REPORT

Received Date: 19 February 2020

Accepted Date: 15 April 2020

Publication Date: 23 April 2020

Clin Exp Ocul Trauma Infect. 2020; 2(1): 20-23

Expulsive Suprachoroidal Hemorrhage During Continuous Positive Airway Pressure Treatment

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Abstract

One of the mechanisms of expulsive suprachoroidal hemorrhage is pressure to the sclera that causes stretching and rupture of posterior ciliary arteries in the presence of full thickness wounds. With this report we present a case of expulsive suprachoroidal hemorrhage in the presence of a full thickness graft–host junction wound.

A 73-year-old female patient had a full thickness rupture of the graft–host junction in the 4 o'clock hour quadrants. Expulsive suprachoroidal hemorrhage occurred shortly after the initiation of continuous positive airway pressure therapy, and light perception was negative. The intraocular tissues were replaced in the globe after the drainage of the intraocular hemorrhage and the existing graft was sutured to the host under sub-Tenon's anesthesia. Although the wounds and sutures remained stable, visual acuity did not improve.

Key words: Suprachoroidal hemorrhage, trauma, penetrating ocular trauma, perforating ocular trauma, continuous positive airway pressure

Introduction

Expulsive suprachoroidal hemorrhage (SCH) is characterized by the expulsion of intraocular structures through a full thickness wound. This usually occurs during intraocular surgeries, especially with large corneal wounds.(1) A rupture of the pos-

terior ciliary arteries resulting from any cause can lead to suprachoroidal hemorrhage. Mechanical pressure to the sclera leads to slight anterior movement of the posterior ciliary arteries that can end with their rupture and the expulsion of intraocular content through a full thickness wound, if one is present. A full thickness wound can occur easily after blunt trauma in an eye that has undergone penetrating keratoplasty (PK).(2)

In this context, we present a case of PK, in conjunction with a full thickness dehiscent graft–host junction due to blunt trauma, in which expulsive SCH occurred a few minutes after the initiation of continuous positive airway pressure (CPAP) therapy for lung edema.

Case

A 73-year-old female with a tiny face had a history of fall or fainting. She was hospitalized for lung edema and pneumonia in the intensive care unit of the emergency department. A PK had been performed for bullous keratopathy 3 years previous, and sutures had been removed 1 year previous to the admission. Visual acuity was 0.5 (in decimal notation) with +5.00 120° 6 months previous, and a mild epiretinal membrane was detected. She was using anti-hypertensives and was not using anti-aggregants or anti-coagulants. Her blood pressure was 132/73 mmHg one hour before the CPAP. The ophthal-

mic examination revealed a full thickness graft-host junction from the 4 o'clock quadrant to the 8 o'clock quadrant, and the graft was displaced anteriorly with slight iris prolapse (Figure 1). The eye was patched, and an urgent primary repair was considered. Thus, according the general principal that we do not treat only the eye but a patient with an open glob injury, the obligatory pre-operative tests and consultations were arranged to sustain a stable medical status for safe anesthesia and postoperative course. A hard eye shield was prescribed, in case the operation could not be arranged due to the patient's systemic problems. After two hours the patient was consulted again for continuous eye bleeding which began a few minutes after CPAP application using an oronasal mask. All of the intraocular structures were protruded from the graft-host junction, and the graft was held in place superiorly, in three-clock-hour quadrants (Figure 2). Visual acuity was perception negative. Informed consent for repair and evisceration were obtained. The operation was conducted straightaway under sub-Tenon's anesthesia due to the high risk for general anesthesia. The primary goal was set to internalize the uveal tissue if possible and to suture the original graft to the host cornea.

Intraocular pressure was high due to blood accumulation in the globe. In order to replace the intra-ocular tissues the hemorrhage was drained with a 21-gauge needle posterior 3.5 mm posterior to the limbus in the superonasal quadrant. After the softening of the scleral cup the uveal tissue was gently loosened from the margins of corneal wound and replaced in the globe. The original graft was sutured with 10-0 nylon in an interrupted fashion. In order to achieve additional support, amniotic membrane was transplanted as a patch from limbus to limbus and sutured continuously with 10-0 nylon (Figure 3). Prophylactic subconjunctival gentamicin and dexamethasone were injected. The patient was calm and cooperative during the operation. Postoperatively, the patient was transferred to the same intensive care unit. Fortified topical vancomycin and ceftazidime were ordered hourly, and dorzolamide-timolol maleat fixed combination, brimonidine tartarate, and preservative-free artificial tears were ordered postoperatively. The eye remained perception negative postoperatively at the third month. The graft was edematous; the graft-host junction remained attached and sutures intact (Figure 4).

Figure 1. A full thickness graft-host junction from the 4 o'clock quadrant to the 8 o'clock quadrant.

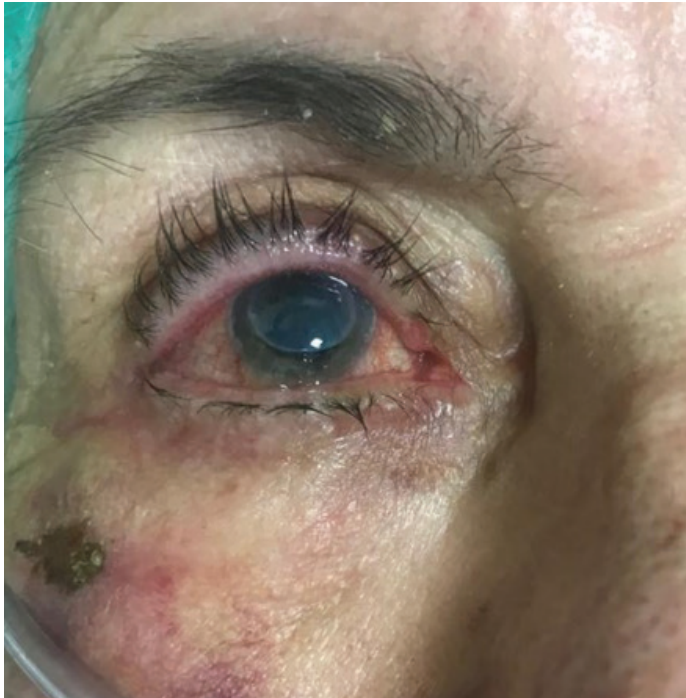


Figure 2. Expulsive suprachoroidal hemorrhage.

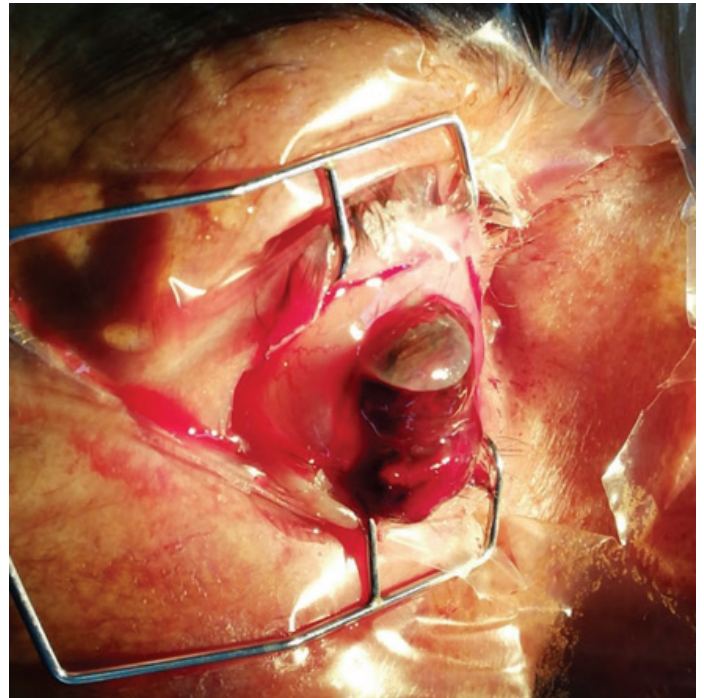


Figure 3. The appearance of the after the operation.



Discussion

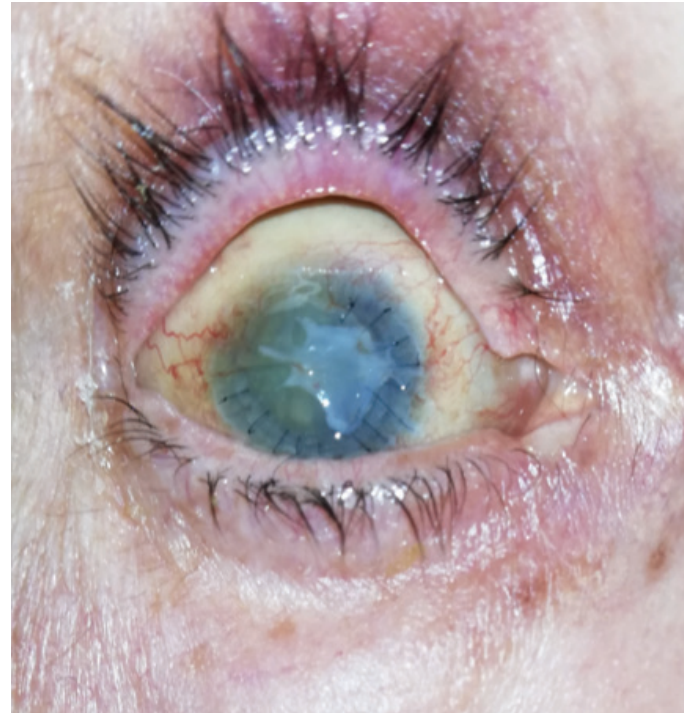
The most common, and the most devastating complication of intraocular surgeries is intraoperative expulsive SCH, and the literature contains many reports on the topic.(3) Less commonly, expulsive SCH can occur in cases with corneal melting or ulcer.(4-6) Although the globe does not contain a full thickness defect, the increased intraocular pressure due to suprachoroidal pressure leads to a rupture of the least resistant point of the globe, such as a thinned cornea, and intraocular tissues protrude through that wound. In this context, we present a case of SCH causing prolapse of the intraocular tissues through a traumatically ruptured PK graft–host junction few minutes after the initiation of CPAP therapy.

Many systemic and ocular risk factors have been determined. Among the associated systemic risk factors including old age, anticoagulant or thrombolytic therapy, systemic hypertension, atherosclerosis, diabetes mellitus, blood dyscrasia, chronic renal disease, and the Valsalva maneuver, our patient had systemic hypertension regulated with anti-hypertensives. (7)

The ocular risk factors are age-related macular degeneration (AMD), glaucoma, and high myopia.(8,9) In experimental studies it has been shown that the strength of the graft–host junction will never be as strong as the cornea.(2) Thus, we think that the main risk factor in our case was the ruptured graft–host junction.

The rupture of long ciliary or posterior ciliary arteries resulting from any cause leads to SCH. When the eye is perforated the

Figure 4. The appearance of the eye three months postoperatively.



intraocular pressure drops to the atmospheric pressure. This breaks the equilibrium and leads to the congestion of choroidal vessels which causes exudation from normal vessels into the suprachoroidal space. Consequently, the suprachoroidal space enlarges and choroid moves anteriorly, stretching the posterior ciliary arteries or long ciliary arteries and disruption of arteries occurs. (1)

Studies have demonstrated that in obstructive sleep apnea syndrome the choroidal thickness decreases(10) and then increases 12 months after the initiation of CPAP therapy.(11) However, we do not think that the current case of expulsive SCH was caused by the effect of CPAP therapy itself; rather, we believe it resulted from the direct mechanical pressure of the oronasal mask on the nasal sclera of our patient, who had a small face, which led to stretching and, consequently, rupture of the ciliary arteries. Increased pressure due to bleeding led to the expulsion of intraocular tissues from the globe through the full thickness defect, further tearing the graft–host junction.

Suprachoroidal hemorrhage usually occurs when the choroidal blood pressure overcomes the intraocular pressure, and it is a devastating complication of intraocular surgeries; especially those with large, full thickness incisions such as PK and extracapsular cataract extraction. However, it is also reported in surgeries with small incisions such as Descemet's membrane endothelial keratoplasty.(12)

The full thickness wounds can be closed in 24 hours. In this case, the characteristics of the wound at the first examination were appropriate for closure in 24 hours. During the closure

of open globe injuries further expulsion tissues must be prevented. Thus, the patient must feel no pain, be immobilized and in stable condition. This can be achieved by general anesthesia which eliminates the need for retrobulbar or peribulbar injection that can elevate intraocular pressure leading to additional tissue expulsion, probable coughing, incoherence and agitation. Thus, when taking into consideration that we don't treat only the eye but the patient, who had pneumonia and respiratory distress, the necessary preoperative testing and consultations were arranged in order to sustain a stable medical status for safe general anesthesia. However, by the expulsion of intraocular tissues which is one of the rare instances that need instantaneous closure, the patient was transported from the intensive care unit to the operating room in the different block of the institution and operated on instantly under sub-Tenon's anesthesia. Fortunately, we were able to close the wound and reposition the intraocular tissues without further ocular or systemic complications. We do not want to create the impression that perforated eyes can be operated upon under local anesthesia; it was obligatory in this case.

In order to drain the hemorrhage we used a 21-gauge needle, as the trocar was not available during the night shift when the operation was performed.

A PubMed literature search did not produce a case of SCH after CPAP treatment. Thus, in reporting this case we want to emphasize that any full thickness rupture or laceration of the globe that cannot be restored by bandage lens or tissue adhesives must be operated upon as quickly as possible. Although we scheduled a prompt repair, the expulsive SCH occurred one hour after the consultation. Another point we wish to highlight is the importance of closing the eye with a hard eye shield prior to the operation, in order to prevent any incidental mechanical pressure which can lead to stretching and rupture of ciliary arteries. Unfortunately, in the current case the expulsion occurred before the eye shield was obtained.

In conclusion, the eye must be repaired as quickly as possible in cases of full thickness corneal or scleral perforation, and the eye must be occluded with an eye shield in order to prevent expulsive SCH.

References

1. Kuhn F, Morris R, Mester V. Choroidal detachment and expulsive choroidal hemorrhage. *Ophthalmol Clin North Am* 2001;14:639-50.
2. Williams MA, Gawley SD, Jackson AJ, Frazer DG. Traumatic graft dehiscence after penetrating keratoplasty. *Ophthalmology* 2008;115:276-8.
3. Savastano A, Rizzo S, Savastano MC, Piccirillo V, Forte R, Sbordone S et al. Choroidal effusion and suprachoroidal hemorrhage during phacoemulsification: intraoperative management to prevent expulsive hemorrhage. *Eur J Ophthalmol* 2016;26:338-341.
4. Oshida T, Kamura Y, Sawa M. Demographic study of expulsive hemorrhages in 3 patients with infectious keratitis. *Cornea* 2011;30:784-6.
5. Martorina M. Spontaneous corneal perforation with expulsive hemorrhage. *Ann Ophthalmol* 1993;25:324-5.
6. Lam A, Faye M, Borzeix A. [Spontaneous expulsive hemorrhage after total necrosis of the cornea. Apropos of 3 cases]. *J Fr Ophtalmol* 1991;14:643-6.
7. Sekine Y, Takei K, Nakano H, Saotome T, Hommura S. Survey of risk factors for expulsive choroidal hemorrhage: case reports. Substantiation of the risk factors and their incidence. *Ophthalmologica* 1996;210:344-7
8. Knox FA, Johnston PB. Spontaneous suprachoroidal haemorrhage in a patient with age-related macular degeneration on excessive anticoagulation therapy. *Eye*. 2002;16:669e670.
9. Chak M, Williamson TH. Spontaneous suprachoroidal haemorrhage associated with high myopia and aspirin. *Eye*. 2003;17:525e527.
10. He M, Han X, Wu H, Huang W. Choroidal thickness changes in obstructive sleep apnea syndrome: a systematic review and meta-analysis. *Sleep Breath* 2016;20:369-78.
11. Uslu H, Kanra AY, Cetintas G, Tatar MG. Effect of Therapy on Choroidal Thickness in Patients with Obstructive Sleep Apnea Syndrome. *Ophthalmic Surg Lasers Imaging Retina* 2018;49:846-51.
12. Dockery PW, Joubert K, Parker JS, Parker JS. Suprachoroidal Hemorrhage During Descemet Membrane Endothelial Keratoplasty *Cornea* 2020;39:376-78.