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Case Report

# Vision Disturbance (not vision loss) following Epinephrine Injection

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**Abstract:**

Perioperative vision loss (POVL) and vision impairments (PVI) following non-ophtalmic surgery are rare, however are serious adverse events of anesthesia and surgery. The outcome of both complications might be blindness or permanent serious vision deteoration. The underlying mechanisms of POVL have been analyzed to a fair level. In contrast, for PVI, they are poorly understood. It is unlikely that POVL and PIV share common etiopathologies. Guidelines for the benefit of patient safety require evidence-based knowledge of the development of complications. To contribute to the knowledge about the causes of PVI, we report a case of PVI following tympanoplasty due to high systemic epinephrine levels.

**Key words:** perioperative vision impairment, perioperative vision loss, epinephrine, hypokalemia

## Introduction

Vision impairment following general anesthesia for a routine surgical procedure is a serious complication. It is unexpected in a field of increasing patient safety associated with anesthetic techniques. Both, anesthesia dependent mortality and morbidity in general continued to decrease since decades despite an increase of risk factors and age of treated patients. Postoperative vision loss (POVL) for non-ophtalmic surgery is rare, however a catastrophic event. The main focus of the American Society of Anesthesiologists (ASA) Postoperative Visual Loss Registry, respectively the ASA Closed Claims Database is on the postoperative loss of vision. Typical surgery such as spine and cardiac procedures, associated factors such as prone position and pathophysiologies such as ischemic opticus neuropathy could been identified (Mendel et al., 2017) and clinical guidelines for the avoidance developed (Lee, 2013; Stambough et al., 2007; Uribe et al., 2012). Compared to POVL, postoperativ vision impairments clearly are alarming events in a similar way. However, PVI is reported even more sparsely. Furthermore, it may be due to other mechanisms than those for POVL. For the avoidance of all vision complications in non- ophtalmological surgery, the attention to the kind and possible mechanisms of this phenomenon is important.

Herewith, we report a case of PVI. We suspect that the intraoperative complication caused the PVI, in this case with good outcome.

## Case

A 27-yr-old male was scheduled for a revision of a tympanoplasty for continuous ear drainage from cholesteatoma. The otherwise healthy outpatient had no history of circulatory diseases, did well during physical exercise, no known drug allergies, uneventful anesthesia history for various smaller surgical procedures under general anesthesia with laryngeal mask airway a year ago. Laboratory values were not requested preoperatively. Vital signs were normal and the patient was slightly anxious but well.

Anesthesia was induced with fentanyl 200 mcg and propofol 200 mg IV in the normotensive calm patient after premedication with 2mg midazolam monitored by 5 lead ECG, non-invasive blood pressure (NIBP) cuff and pulse oximetry. Endotracheal intubation was accomplished after administration of rocuronium 50 mg. Maintenance of

anesthesia with 3vol%-5vol% sevoflurane given in 60% air / oxygen mixture was induced during a period of vital parameters check, auscultation of bilateral ventilation, tracheal tube positioning and securing, eye taping, nasopharyngeal temperature probe positioning and surgical skin preparation. Antibiotic prophylaxis was 1g cefotan i.v. The surgeon injected 5 mL admixture of local anesthetic with epinephrine into the premastoidal skin and close to the skull base and saw the typical skin paling. As soon as he terminated this latter procedure of approximately 3 –5 minutes, heart rate increased from sinus rhythm 62/min to Vtach with 198 min-1 max. 40 mg lidocain were injected for successful conversion to sinus tachycardia with 178 max. NIBP increased to 225 over 135 mmHg maximal. Esmolol was titrated (150 mg total) to reduce heart rate and blood pressure, desflurane and oxygen delivery was increased to 7.2 Vol% and an inspiratory fraction of oxygen of 0.99, respectively. Both pupils were maximal dilated, the ipsilateral pupil slightly contorted. Surgical proceeding was canceled. Blood analysis showed hypokalemia of 2.6 mEq $L^{-1}$, Na 138 mmol $L^{-1}$, Cl 101 mmol $L^{-1}$, CO2 25 mmol $L^{-1}$, Phosphorus 37 mg $dL^{-1}$, CK 73 mg $dL^{-1}$, Troponin I <0.3 mcg $mL^{-1}$, euthyroid TSH, pH 7.32. As soon as the vital parameters had been back to normal, extubation and transfer to the recovery room was possible, 38 min after start of arrhythmia. Although the alert patient did not complain about chest pain or shortness of breath, he received 6 L min-1 oxygen via a facemask, IV nitroglycerine and potassium chloride. Hypotension was treated with phenylephrine. Pupil size and form also recovered within 2.5 hours, associated with considerable photophobia, improving in parallel. Also, he complained about after-images. Without further events, he was monitored the following night. Besides hypoproteinemia (total protein was 5.3 g $dL^{-1}$, albumin 3.0 g $dL^{-1}$), hyperglycemia 160 mg $dL^{-1}$, and hyperbilirubinemia (1.3 mg $dL^{-1}$), other routine values including CBC were normal. Troponin I level rose within 6 hrs to 16.2 ng mL-1 but declined to 12.6 after 10 hrs after the event. Visual distortion and metamorphosia was the only subjective complaint in the morning. The patient was discharged from the cardiology unit the following day.

The history of the patient was taken again on postoperative day one, with specific concentration on a cause for potassium loss. He did not take diuretic drugs or sympathomimetic inhalers, but had a short period of diarrhea, lasting until 7 days prior to surgery. During diarrhea and until the day of surgery lightheadedness and dizziness occurred almost daily.

Fourty-eight hours after the event, the patient complained about blurred and cloudy vision together with problems to focus, decreased distance vision, waved lines but denies headache, diplopia, or blind spots. Ophtalmological counsil reported normal visual acuity 20/30 R and 20/40=1 L, improving with manifest retraction to 20/25-1 R and 20/25/-2, intact confrontational visual fields, intact pupil reaction to light without papillary defects, intact extraocular movements, mild exophoria, normal slit lamp exam. Furthermore, stereo vision test showed good depth perception with 9/9 correct Titmus circles. Fundus exam was normal, also, no hemorrhages, no exudates, no cotton-wool spots. Goldman or Humphrey visual field was not performed as scotoma were denied and confrontational visual fields were full. In conclusion, mild myopia and astigmatism as well as mild exophoria were likely been present before surgery. His problems to focus, however, did vane 10 days after and the aquarium like vision after 18 days.

He was rescheduled for the surgical procedure 3 month later and had uneventful anesthesia and surgery including a recovery phase without vision impairments.

## Discussion

This case of PVI was related to a specific intraoperative event, i.E. high dose epinephrine local injection in the neck region. Therefore, we are able to relate the uncommon symptoms to a rarely observed pathophysiology. This demonstrates the need for increased attention to all kind of vision impairments following non-ophtalmological surgery.

To date, our knowledge about perioperative vision changes is limited- as it is for POVL (for recent reviews see (Roth, 2009) (Lee, 2013; Newman, 2008; Shmygalev & Heller, 2011)). The phenomenon of permanent vision impairment after anesthesia for major surgical procedures, particularly cardiopulmonary bypass or spine surgery may defer from the incidence of POVL since PVI is not published frequently. Permanent perioperative vision loss following spine and cardiac surgery primarily is due to ischemic optic neuropathy (ION) caused by extended anesthetic duration (>5h), blood loss over 1L, Mayfield pins, or bilateral disease (Kamming & Clarke, 2005). Only to a much lesser degree, the etiology is central retinal arterial or vascular occlusion (RVO) (Lee et al., 2010; Lee et al., 2006). In cardiac surgery, 0.06% of patients suffer perioperative vision loss for not exactly known reason (assumed are higher age and extensive comorbidity, procedural characteristics (extracorporal circulation, cardiovascular trauma, emboli), and profound hypotension) (Shaw et al., 1987). In non-cardiac surgery, vision loss seems to be caused by intraoperative anemia, prone position and sustained compression of the globe, hypotension, prolonged surgery duration resulting in ischemic optic neuropathy, central retinal artery or vein occlusion, or cortical blindness (data originating from the American Society of Anesthesiologists (ASA) Postoperative Visual Loss Registry, respectively the ASA Closed Claims Study). Consequently, practice guidelines have been developed to avoid this kind of complication (Loss, 2019).

However, slighter ocular complications as changed vision, refraction alterations, scotoma are not to be reported. The question erases, if the same mechanisms for vision loss and “minor” ocular complications are responsible. Thus, the mechanisms assumed to be responsible for POVL should be considered in more detail: Five categories have been charged: External ocular injury (corneal abrasion or sclera injury), cortical blindness, retinal ischemia, ischemic opticus neuropathy (ION) and subtypes, and acute glaucoma (Grover & Jangra, 2012). However, the presented case does not fall into all of these categories. Instead, epinephrine overdose from local anesthesia injection is the most possible reason for the transient vision disturbance after anesthesia. Iatrogenic vasoconstrictor injections during anesthesia increased normal to assumable high plasma epinephrine levels, possible resulting in short generalized vasoconstriction systemically. The vasoconstriction of arachnoidea and ocular arteries might have produced reversible ischemic edema, likeliest of the arachnoidea since all ophtalmological evaluations were without changes. Taken from several old sources like the Oregons National Registry of Drug-Induced Ocular Side Effects, ocular effects of systemic epinephrine plasma levels include mydriasis, color vision defects (green tinge to objects), hemianopsia, acute macular neuroretinopathy (AMN). The latter, acute macular neuroretinopathy, was reported from independent groups (Desai et al., 1993; O'Brien et al., 1989). Following intravenous sympathomimetics, patients suffered from central vision loss, paracentral scotomas, early macular edema, macular red spots, later red diffuse macular lesion, as well as persistent scotoma for 6-8 weeks after the episode. Our patient might have had AMN, but this is unlikely given no fundus findings and no paracentral or central scotomas on Amsler Grid). Although, he did have nearly immediate symptoms like these patients did the same. In contrast to the described symptoms, he reported a fish bowl appearance and waviness of his vision

instead of scotoma. One may assume that vision effects following intravenous sympathomimetics are seen more frequently in patients after resuscitations and long-term ICU catecholamine treatment but reports from these fields are missing in common data bases. One group reports various affections of the optical system including bilateral retinal artery and choriocapillaris occlusion following the injection of epinephrine or lidocaine together with long-acting corticosteroid suspensions which either produce direct neurotoxic effects or embolic phenomena (McGrew et al., 1978; Wilson et al., 1978). Another possible reason for that kind of reported vision impairment is perioperative drug use (central sympathomimetic agents such as 3,4-methylenedioxy-methamphetamine (MDMA, "ecstasy"), pseudoephedrine, and others can cause palinopsia and polyopia (after images, ghost or echoed images) through central serous chorioretinopathy (McGuire et al., 1994; Passie et al., 2002). However, both reasons do not apply for our case. The reason for the extended visual impairment in this case might be either a prolongated effect of the epinephrine to the mechanism of accommodation when the effect to the pupil has already disappeared or a temporary increase of the lens thickness caused by a disturbance of the blood electrolyte balance. There is some evidence in clinical practice more than in literature that the impairment towards accommodation lasts longer than the medical mydriasis caused by sympatomimetic drugs like epinephrine. On the other hand changes in the blood electrolyte balance, especially with hyperglycaemia lead to an increasing level of plasma osmosis thereby creating ocular hypotension, a myopic change in refractive error, narrowing of the anterior chamber and a thickening of the lens (Furushima et al., 1999). A decrease of plasma glucose level will normalize the intraocular pressure and reverse the myopic changes of the eye. It is unlikely that neuropathy of the optic nerve was the reason for the visual disturbances because no afferent papillary defect was detected at any time during the ophthalmologic examinations

Given the absence of morphological findings during ophtalmological exams of the reported patient, central effects on the optical cortex may be involved since pressure-mediated passage through the blood-brain barrier of epinephrine is discussed (Abdul-Rahman et al., 1979).

## Conclusions

Perioperative transient vision impairment (PVI) is probably due to other mechanisms than those for POVL. In the reported case it might be caused by central effects of local epinephrine injections in the neck region or by transient changes of intraocular structures especially intraocular pressure and lens thickness.

### Disclosure of Interests

No Disclosure of interests for all authors

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